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# ANATOMY OF INVERSION AND RECESSION OF IRIS.

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The anatomy of such lesions is described, and the mechanical conditions under which they are produced are discussed. The explanations of these are illustrated by typical cases, from the literature, or from the Eye Clinic of Professor J. Meller of Vienna.

Since the first description of iris inversion and recession by Schmidt (1805) and Ammon, these subjects have always aroused interest among ophthalmologists. Recently Groenouw[1] published six such cases.

Inversion and recession of the iris can be observed rather frequently clinically, tho it is often overlooked. In the following I will give a description of a few additional cases, together with the mechanical principles involved. The accompanying illustrations are sketches made with the camera lucida, and therefore, represent the true relations of the anatomic details.

I wish to state in advance, that in inversion the iris lies completely back upon the ciliary body, whereas, in recession the iris is folded and incarcerated between ciliary body and lens. Clinically, in cases of inversion we can see nothing of the iris at the point of inversion, whereas in recession, we can often see it greatly narrowed at this point. I wish here to presuppose that recession represents a slight degree of inversion, and is often a transitional stage between an inverted iris and the normally placed iris.

IRIS INVERSION FOLLOWING OPERATIONS.

Clinically, we find inversion and recession of the iris after cataract opertions in which prolapse of the vitreous has occurred. This is especially the case when operating with the round pupil, as is the rule in linear extraction. When vitreous follows the expression of the nucleus, the iris which lay in the wound during the manipulation disappears, the anterior chamber is restored by the vitreous; and the pupil is drawn upward. If the vitreous is fluid, the wound can be absolutely closed. The vitreous which prolapsed into the anterior chamber has pushed the iris backwards everywhere, thus restoring the depth of the anterior chamber. In the region of the wound, the iris has been pushed especially far back, being pressed against the ciliary body and, therefore, the iris is not seen in the wound. The iris is then often fixed on the ciliary body and later the pupil remains retracted upwards, with the two limbs of the apparent coloboma running back into the depth of the eye.

Thus the iris often disappears, in cases where the lens was extracted with prolapse of the vitreous. But this happens more especially in attempting an iridectomy, in cases where the lens was luxated by trauma for instance, and the anterior chamber was filled with vitreous. Here the anterior chamber becomes deep after the incision due to the presence of vitreous. The iris drops so far backwards, especially in the region of the incision, that it is often impossible to grasp it with the forceps for the purpose of making an iridectomy. It is scarcely possible to thoroly excise a piece of iris, even in case one has been fortunate enough to grasp it with the double-curved Hess iris forceps; because the vitreous continually presses it backwards.

In the following, I will quote a case as an example, (Fig. I). Three months previously a discission had been made on the left eye of a fourteen year old girl, on account of a dense perinuclear cataract. The swelling of the lens stopped after a while, and it was decided to perform a linear extraction. The operator who had performed the

first operation assured me that he had not injured the posterior capsule in making the discission.

Operation: Dec. 3, 1924. A large piece of capsule is removed with the capsule forceps, thru a moderately large keratome incision in the lower

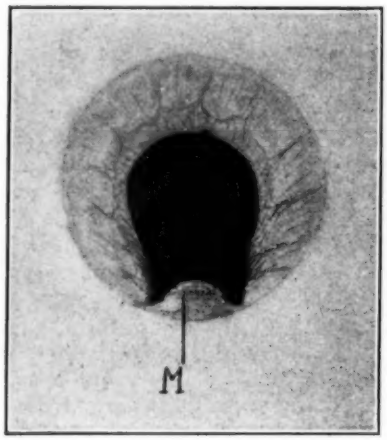


Fig. 1.—Appearance of pupil after second operation for juvenile cataract. Recession of lower part of iris, the mass M, an apparent coloboma, is composed of opaque vitreous and lens remains.

part of the limbus. During the massage some vitreous follows the lens matter. A black hole appears in the iris, the anterior chamber becomes deep and the pieces of lens matter are driven to the periphery of the anterior chamber. The wound does not gape, indeed is firmly closed. The lower part of the iris has disappeared, but does not lie in the wound. It is impossible to replace the iris with the spatula.

The eye healed without complications. The sketch (Fig. I) was made fourteen days after the operation. The lowest part of the iris is lacking, the margins of the iris disappearing into the depths of the eye. In the region of the supposed coloboma lies a gray mass (M) which is composed of opaque vitreous and pieces of lens matter. Here at the bottom, the iris is inverted and apparently lies on the ciliary body. The wound is firmly closed and nothing can be seen of an inclusion of the iris.

The vision is 6/8 after two months, and the clinical picture is slightly altered. The iris margins no longer run into the depth of the eye, but now are drawn to the wound. The mass of tissue (M) in the lower angle of the chamber has apparently shrunken and pulled the iris up towards itself.

IRIS INVERSION AFTER INJURY.

Inversions and recessions are less frequent in cases of injury. All partial iris inversions which I had the opportunity to examine anatomically were found in cases of perforating injuries. They therefore do not quite correspond to the clinical picture described by Ammon[2]. In his cases, the skull had been shattered by a shot, and the iris had disappeared in an uninjured bulbus. This was clinically visible as the instantaneous death of the patient had apparently prevented any hemorrhage.

Iris inversion occurs in perforating injuries when the wound lies in front

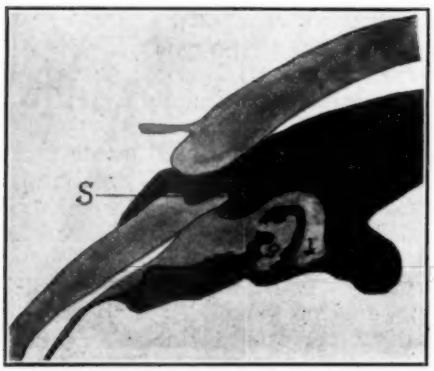


Fig. 2.—Rupture of eyeball near limbus. S, scleral spur; I, iris pushed backward upon ciliary body.


Fig. 2a.—Another section from the same eye showing also laceration of the ciliary body.

of the scleral spur as well as when it lies back of it. In regard to the first group, one finds the majority of iris inversion in typical indirect scleral ruptures. Such a case is shown in Fig. 2. The rupture lies just in front of the scleral spur (S), and runs obliquely backwards and outwards; the ciliary body has been torn inwards slightly, and the iris (I) has been thrust backwards, and lies upon the ciliary body. Blood lies in the wound and anterior chamber.

We have before us therefore, a case of iris inversion. In the neighboring sections,[3] the iris does not lie spread out over the ciliary body, but lies folded on the ciliary processes (Fig. 2a). Here at the front, the ciliary body is torn inwards still more, and is spread out, while the iris (I) is folded at an acute angle. In this recession, we have apparently a transitional form between the complete iris inversion and the normal position of the iris. Probably many of the iris recessions demonstrated anatomically are only such folds and transition forms between the normal position and complete inversion of the iris.

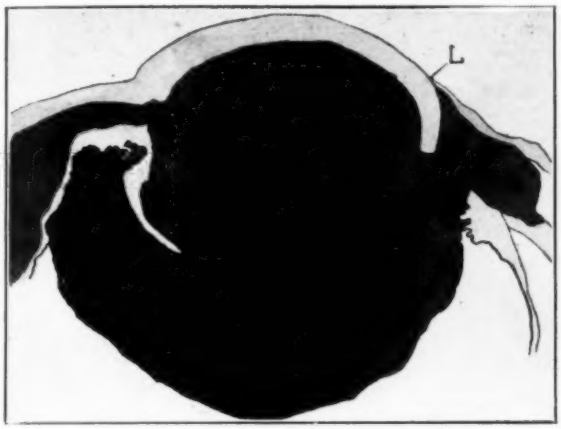


Fig. 3.—A gaping scleral rupture behind limbus L.

The mechanism of this displacement can probably be attributed to the prolapsing vitreous which, following the escaping lens, breaks the hyaloid membrane in the fossa patellaris, enters the anterior chamber, and presses

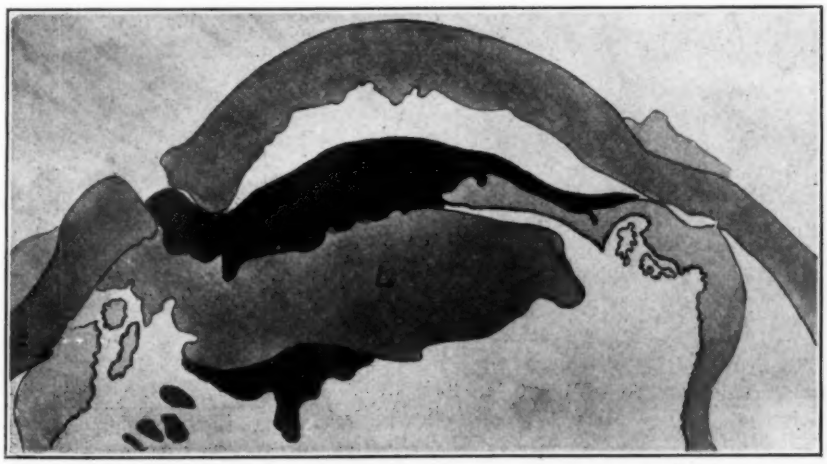

Fig. 4.—Rupture of cornea. S, scleral spur; L, remains of lens.

the iris backwards. A similar explanation is given by Groenouw.

INVERSION AND RECESSION OF IRIS.

In a similar manner blood seems to be able to press the iris back. In Fig.

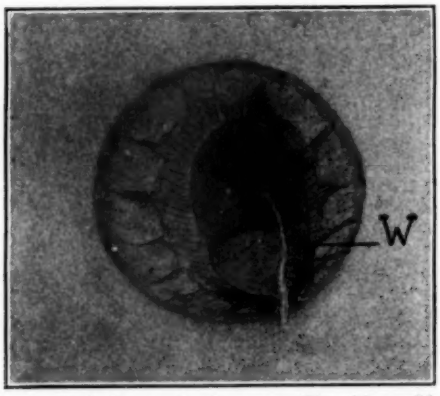


Fig. 5.—Incised wound of cornea, W, with pushing back of iris below. Appearance three days after injury.

3, we see a gaping scleral rupture. The perforation lies in front of the scleral spur, and runs obliquely thru the sclera. It is far back of the limbus (L) which is recognized by means of the tab of conjunctiva. The iris is lacking at this point, having been expelled with the lens by the force of the injury. The ciliary body on the other side is detached, and the iris is thrust far backwards and lies against the ciliary body. Blood fills the anterior chamber, the subchoroidal space, and part of the vitreous cavity. In this case the iris inversion is seen on the side opposite to the point of injury.

In a third case, (Fig. 4), the classical explanation of Foerster, as given in the text book of E. Fuchs (Duane),[4] might well be the proper one. Here the perforation lies in the cornea, far in front of the scleral spur (S). The cornea is folded and more curved, as is typical in such injuries, so that the central edge of the wound is deviated toward the center. The anterior chamber is filled with blood, and the lens (L) injured, and lens matter is lying against wound. The iris is absent on one side, and the ciliary body has slid forward. On the other side the iris is pressed back into the circumlental space in an acute angle. So here also the recession, which could not be seen clinically on account of the hemorrhage, lies on the opposite side from the injury.

Recently I was able to observe clinically a case belonging to this group (Fig. 5). A three year old boy had been wounded in the eye with a knife three days previously. A perforating incised wound (W) runs from the middle of the cornea to the lower limbus. The wound is sealed with fibrin. The iris is neither incarcerated nor prolapsed. The iris becomes very narrow in its lower part and runs towards the back. One can clearly see that the iris is not connected with the wound. The lens is cloudy and parts of the lens capsule are appar-

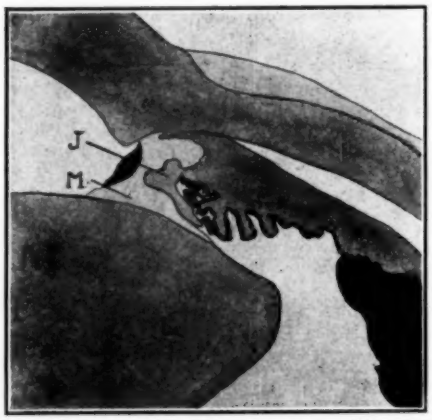


Fig. 6.—Injury of eye near equator. Hemorrhage on posterior portion of ciliary body. J iris resting on ciliary body in circumlental space. M, connective tissue membrane; L, lens dislocated.

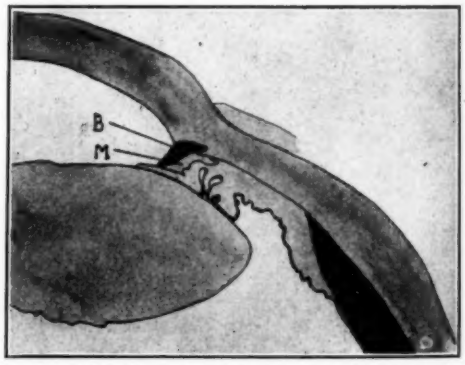


Fig. 6a.—Neighboring section, iris folded. B, blood; M, membrane.

ently connected to the wound. Laterally there is a posterior synechia. It was impossible to examine this case with the slitlamp, on account of the unruliness of the child. There is no doubt, however, that here, too, vitreous had prolapsed into the anterior chamber and had pushed the lower part of the iris back.

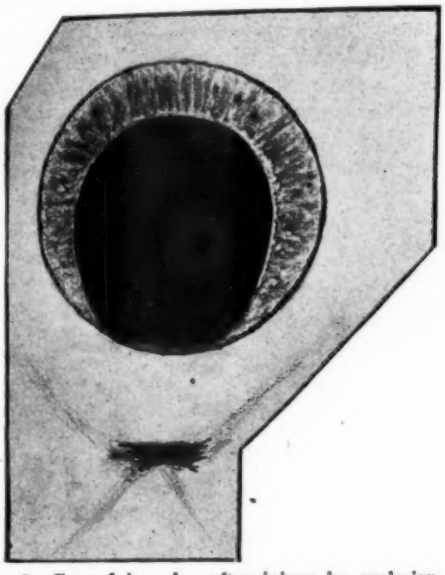

Fig. 7.—Eye of boy day after injury by explosion. Large hole in sclera below Anterior chamber very deep; iris disappeared behind lower limbus.

The perforating wound can also lie *behind the scleral spur*. In this case the iris inversion or recession seems to lie on the same side as the injury. Here also we found in one case how the recession can be a transition form of complete inversion.

Fig. 6 is from a case where the injury was at the equator, and so does not show in the sketch. One only sees the dark blood lying on the flat portion of the ciliary body, to which it had spread from the point of injury. The iris (J) has become necrotic and shrunken, due to the trauma, and lies spread out upon the ciliary body in the circumlental space. Upon it lies a connective tissue membrane (M), which fills in the space between lens and the angle of the anterior chamber. The lens (L) is luxated and lies pressed against the ciliary body. Fig. 6a, shows a neighboring section of the same eye. Here the iris is not thrust back, but merely folded. The connective tissue membrane (M) lying upon it is much


Fig. 8.—Section of eyeball perforated behind scleral spur. B, blood; P, pigment line in fold of iris; R, displaced retina.

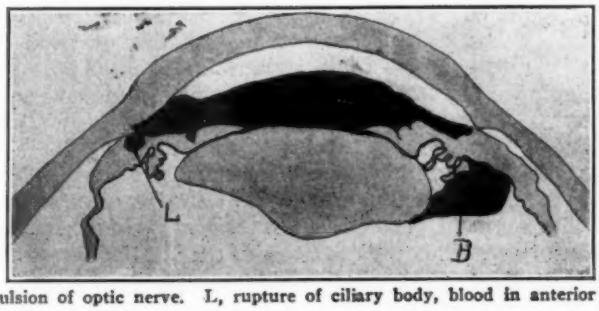


Fig. 9.—Case of evulsion of optic nerve. L, rupture of ciliary body, blood in anterior chamber and at B.

smaller, and is covered by a small layer of blood (B), (colored dark in sketch). Here the lens is thrust still further against the ciliary body.

In another eye I was able to obtain quite similar findings. Recently I was also able to see a similar case clinically (Fig. 7). This was an eight year old boy whose left eye had been injured the day before by a flying piece of brass in an explosion. In the region of the equator, below, there was a rather large hole in the sclera, black, and filled with vitreous. Four shallow grooves in the

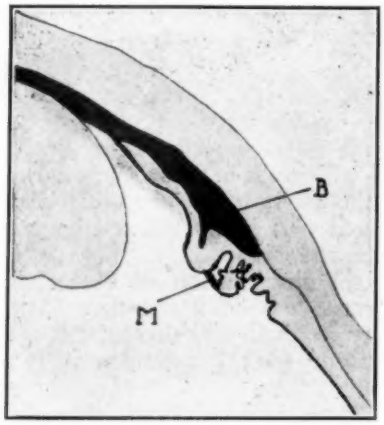


Fig. 10.—Iris drawn back by membrane M. Anterior chamber filled with blood B.

sclera ran to this hole. The cornea was brilliant and transparent. The anterior chamber was very deep. The iris disappeared behind the limbus in its lower part, and the anterior chamber became definitely deeper in this region.

This case represents, apparently, the section shown in Fig. 6.

A further case belonging to this group is sketched in Fig. 8. The perforation lies immediately behind the scleral spur. A part of the ciliary body and choroid were expelled with the lens. The cornea is curved more sharply than normal, and the posterior surface is thrown into folds, which appeared clinically as striate opacities of the cornea (keratitis striata). The iris is folded on the side of the wound and hangs on the scleral spur. The fold resulting from this recession is completely filled with pigment cells (P). These cells come from the pigment epithelium. They have separated from the iris, which has become partially necrotic due to the severe trauma, and after entering the anterior chamber, have been washed into this fold in the iris. The wound is filled with blood (B) and behind it a piece of the retina (R) is visible.

This kind of iris inversion in injuries perforating posterior to the scleral spur I believe can be explained thus: The foreign body indents the sclera to a considerable degree before perforating


Fig. 11.—Case of scleral rupture, iris bound to ciliary body by synechia S. Prolapse of ciliary body at C.

it, thus suddenly raising the intraocular pressure. This stretches the cornea greatly. After the perforation the intraocular pressure sinks markedly at the point of injury, and the whole contents of the eye rushes towards the opening in the sclera. The aqueous in attempting to reach the wound can depress the iris backwards, or invert it on this side. The lens also can be luxated towards the wound or even be entirely expelled.

A similar explanation probably holds true for the original iris inversion of Ammon. The posterior pole of the eye was suddenly and forcibly compressed by the shattering of the skull. At the sudden release of the increased pressure a recoil followed, due to the elasticity of the anterior part of the sclera and especially of the cornea, and the receding aqueous thrust the iris back between the lens and ciliary body.

This explanation is also supported by the case of Bachstez.[5] In a case of an attempted suicide, the bullet had crossed the region of the chiasm and one eye had been momentarily compressed from the back. The cornea became cloudy, and was protruding, so the eye had to be removed. Anatomic examination revealed, in the upper part of the eye, a recession of the iris and a necrosis of the cornea with lacerations of the external corneal lamellae. The necrosis in this case, apparently,

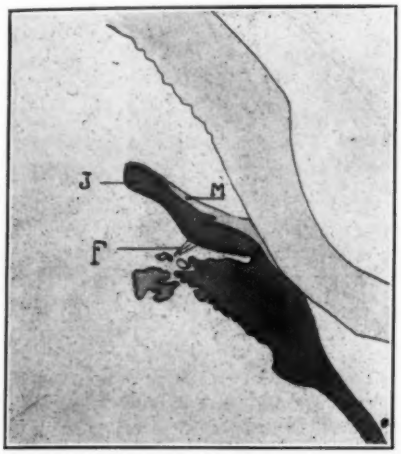


Fig. 12.—M, membrane; J, iris; F, fibroblasts drawing on iris.

followed the compression of the contents of the eye and the stretching of the bulbus. The recoil then caused the iris recession. The case sketched in Fig. 9, also supports this view. The optic nerve had been torn out of this eye, by a piece of hand grenade. We see the anterior chamber full of blood and the ciliary body torn inwards from the angle of the chamber (L), which we so often find in cases of blunt injury. The iris is folded on both sides, especially on the right. At this point, there is also some blood (B) on the flat part of the ciliary body.

IRIS FOLDINGS DUE TO OTHER CAUSES.

The diagnosis of histologic pictures of iris inversion is, however, often very difficult, as Groenouw has previously stated, because different circumstances can produce similar anatomic pictures. In the following paragraphs, I will detail several cases which demonstrate. not true iris inversion, but foldings of the iris, due to other causes.

Thus in Fig. 10, we see a membrane (M), which reaches out from the ciliary body to the posterior surface of the iris, and has drawn the latter backwards. The anterior chamber is filled with blood (B). In another case (Fig. 11), the ciliary body and the choroid had been detached by a large hemorrhage, and the posterior surface of the iris was bound to the flat portion of the ciliary body by a synechia (S). This was a case of scleral rupture with prolapse of the ciliary body (C) and expulsion of the lens.

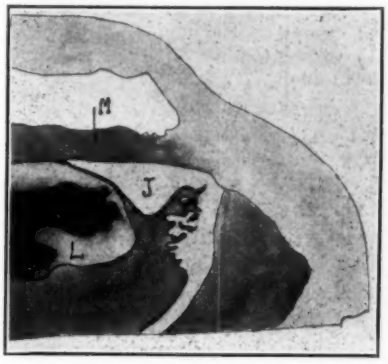


Fig. 13.—Section of shrunken eyeball. M thick membrane; J section of iris; L, calcified lens.

Sometimes the contraction of the granulation tissue on the posterior surface of the iris is reinforced by membranes on the anterior surface; and this, in light cases, leads to foldings of the iris. Thus we see in Fig. 12, a membrane (M) on the anterior iris (J) surface, which is attempting to pull the iris towards the angle of the chamber. The posterior surface, on the other hand, is drawn towards the ciliary body by the contraction of the granulation tissue. Thus the iris (J) is folded, due to the antagonistic action of the traction of the membrane (M) on its anterior surface and the pull of the fibroblasts (F) on its posterior surface.

A membrane can also run across the whole anterior chamber, and be very strong. In such a case it can, by its contraction, press the iris completely into the circumlental space, as in Fig.

13. In this phthisical eye a thick membrane (M) crosses the anterior chamber. The iris (J) shows a triangular cross section. It is not really folded as in recession but rather pressed into the circumlental space. Behind the iris lies another membrane. The lens (L) is calcified.

was increased by the choroidal detachment, which is due to the fixing.

Clinically one sees similar pictures after luxation of the lens following contusions, or after loss of vitreous following perforating wounds of the sclera. The iris draws directly back from its point of insertion, and forms

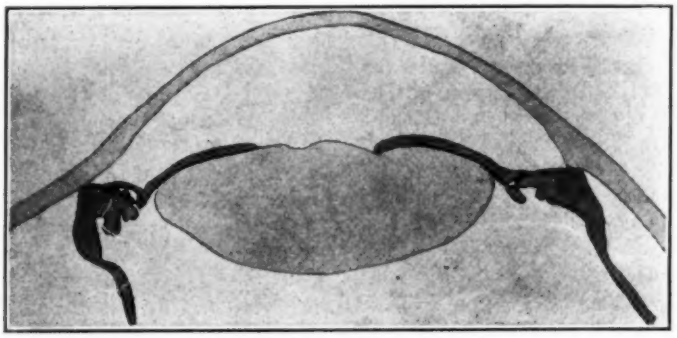

Fig. 14.—Recession of iris due to adherence to lens, subsequently drawn backward.

Fig. 14, shows a very peculiar form of iris recession. The iris is completely adherent to the lens on its pupillary margin. The retina is detached. The choroidal detachment is an artefact. The recession of the root of the iris is much more pronounced on one side than on the other. This can probably be explained as follows: Originally the anterior synechia was formed. Later, due to the retinal detachment, the anterior chamber became deep again. The iris was then proportionately too long, and the peripheral parts folded themselves back into the circumlental space. Perhaps the folding of the iris a "step" in reaching the anterior surface of the lens. In case of a slightly pigmented iris, it is even possible to see the individual ciliary processes thru the sunken iris. This "step" in the iris disappears after a few days, when the loss of vitreous is replaced by fluids, and the anterior chamber has become shallower.

The causes and the modes of development of iris inversion and recession are, therefore, many. The diagnosis from anatomic specimens is very uncertain, when serial sections are lacking; because, as has been shown above, inversion or a recession can be simulated by other pathologic conditions.

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# THE ANTIPNEUMOCOCCIC PROPERTIES OF ETHYLHYDROCUPREIN HYDROCHLORID (OPTOCHIN) IN VIVO.

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Ethylhydrocuprein was injected into the eyes of rabbits to test its power of inhibiting the growth of pneumococci in the living eye. Such injections in the vitreous destroy the function of the eye, so that the pupil will not react to light but do not prevent the growth of pneumococci. In the anterior chamber they cause atrophy of the anterior layer of the iris without other harm; and 0.2 c.c. of 1:100 solution inhibit the growth of the pneumococcus. The experiments were made in the Department of Ophthalmic Pathology in Harvard Medical School under Professor F. H. Verhoeff, and in the Massachusetts Eye and Ear Infirmary.

Morgenroth and Levy[1] in 1911, introduced the base ethylhydrocuprein (optochin) $C_{19}H_{22}N_2OHOC_2H_5$ a derivative of hydroquinin $C_{19}H_{22}N_2OHOCH_3$ which is reduced or hydrogenated quinin obtained from cinchona bark or the laboratory. They found the drug to have a parasiticidal effect on the pneumococci in vitro. In 1915 Wright[2] showed that the action of ethylhydrocuprein hydrochlorid on pneumococci in vitro is only slightly lessened by the presence of serum. Fugendreich and Russo[3] showed that the hydrochlorid of the drug in a concentration of 1 in 16000 kills the pneumococci in vitro in 3 hours at room temperature, and that it is much more potent in this respect than the hydrochlorid of homologous compounds or that of quinin. Moore[4] has shown that the base inhibits the growth and kills pneumococci in vitro in considerable dilutions, and that it exerts a considerable protective action in experimental pneumococcal infections as well as in the case of type strains of all groups of pneumococci. Moore[5] later showed that the serum of rabbit and man which had been previously treated with a single dose of the drug exerts a bactericidal action on, and later inhibits the growth of pneumococci in the test tube. In case of subcutaneous injections of the drug in oil, the maximum effect is about one hour after the administration and lasts about four hours.

However, such injections proved not to be entirely devoid of danger especially as it concerns the eye. Abelsdorff[6] compiled 50 cases of amblyopia due to ethylhydrocuprein. He found that the cause of the amblyopia was similiar to that of quinin, that is, we get a narrowing of the vessels with secondary optic atrophy. There may also be white patches in the retina with sclerosis of the vessels in these areas. The latter is peculiar to ethylhydrocuprein.

With the above facts in mind it occurred to us that optochin should likewise destroy pneumococcal infections in the aqueous and vitreous if the drug was injected directly into these chambers in sufficient strength to inhibit the growth of the pneumococcus.

The present work was undertaken to ascertain if ethylhydrocuprein could be injected into the anterior chamber or the vitreous in sufficient strength to inhibit a pneumococcus infection, without destroying the ocular tissues.

For obvious reasons it was necessary to resort to animal experimentation to determine those points. The eyes of rabbits were used exclusively for this work. Since Cheney[7] showed that type 2 pneumococcus was the most common offender in hypopyon ulcers this type of organism was used exclusively.

The pneumococci were cultured in Avery media and incubated at 98° F. for 8 hours. All cultures were examined microscopically to determine if the culture had grown successfully and also to determine if the culture had been contaminated by other organisms. The optochin was prepared fresh from a concentrated solution diluted with normal saline. Before making the intraocular injections, the anterior chamber was partly evacuated with a hypodermic syringe. No other medication was used in any case.

## EXPERIMENTS.

July 6/22 O. D.—Injected 0.25 c.c. of 1:500 solution of optochin in the vitreous. 7/8/22—Pupil does not react to light. Aqueous clear, iris congested, vitreous cloudy, unable to see

fundus, opacities on posterior of lens. 9/15/22—Opacities on posterior of lens and vitreous gradually cleared. Pupil dilated 5 mm. and does not react to light. Slight opacity on posterior of lens. Vitreous clear, optic nerve very pale, vessels narrow. Vision seems to be nil.

No. 2. July 8/22 O. D.—Injected 0.25 c.c. of 1:500 solution of optochin in vitreous. 9/5/22—Condition and outcome same as in experiment No. 1.

No. 3. August 21/22 O. D.—Injected 0.12 of 1:1000 solution of optochin in vitreous. 8/22/22—Pupil dilated 5 mm. and does not react to light, opacities on posterior of lens and opacities in the vitreous, unable to see the fundus. 9/15/22—Opacities in the vitreous, unable to see fundus. 10/15/22—Opacities on posterior of the lens and in the vitreous gradually cleared. Pupil 5 mm. and does not react to light. Vitreous clear. Optic nerve pale, vessels narrow. Vision appears to be nil.

No. 4. Aug. 21/22 O. D.—Injected 0.25 c.c. of 1:1000 of optochin in the vitreous. 8/22/22—Pupil 3 mm. and does not react to light. Opacities on posterior of the lens and in the vitreous. Unable to see the fundus. 9/16/22—Opacities in the vitreous and on the posterior of lens gradually cleared up. Pupil dilated 3 mm. and does not react to light. Vitreous nearly clear. Optic nerve white, very narrow vessels. Vision seems to be nil.

No. 5. October 15/22 O.D.—Injected 0.3 c.c. of 1:100 solution of optochin in anterior chamber; precipitates immediately formed. 10/16/22—Marked iritis, aqueous cloudy, pupil contracted and does not react to light. 11/14/22—Inflammation of iris gradually cleared up. Eye white and quiet. Pupil reacts well to light. Lens clear. At the point of injection the anterior part of the iris is atrophic, causing a discoloration of the iris, no synechiae present.

No. 6. October 7, 1922 O. D. Injected 0.15 c.c. of 1:100 solution of optochin in the anterior chamber. Precipitates immediately formed in anterior chamber. 10/9/22—Aqueous clear, mild iritis, pupil does not react to light. 11/16 O. D.—Iritis gradually cleared and eye now appears normal except for a slight atrophy at the point of injection with resulting discoloration of the iris.

No. 7. October 7, 1922 O. D.—Injected 0.3 c.c. of 1:100 solution of optochin in anterior chamber, which immediately formed a marked precipitate. 10/8/22—Precipitate in anterior chamber, marked iritis, pupil very small and does not react to light. 11/12—Eye gradually cleared. At present eye appears normal, except for an area of atrophy of anterior layers of the iris at the point of injection which causes a discolorization of the iris.

No. 8. October 7, 1922 O. D.—Injected 0.4 c.c. of a 1:500 solution of optochin in anterior chamber. Precipitates immediately formed. 10/8 O. D.—Slight precipitates in anterior chamber, slight congestion of iris. Pupil fairly well dilated but does not react to light. 11/10 O. D.—Congestion rapidly cleared up. Eye normal except very slight atrophy of the iris at point of injection causing a scarcely perceptible discoloration.

No. 9. September 13, 1922—Injected 0.02 c.c. of pneumococcus culture in anterior chamber. 9/15/22—Marked exudate in anterior chamber. Marked inflammation of the anterior of the eye. Developed panophthalmitis followed by phthisis bulbi.

No. 10. September 16, 1922 O. S.—Injected 0.02 c.c. of pneumococcus culture in the vitreous. Twenty minutes later injected 0.3 c.c. of 1:1000 solution of optochin in the vitreous. 9/17/22 O. S.—Chemosis of conjunctiva. Iris congested, pupil small and does not react to light. Vitreous very cloudy. 9/20/22—Panophthalmitis, condition became worse and ended in phthisis bulbi.

No. 11. September 29, 1922 O. D.—Injected 0.01 c.c. of pneumococcus culture in the vitreous, followed 20 minutes later by 0.2 c.c. of 1:100 solution of optochin in the vitreous. 9/30/22—Marked chemosis of bulbar conjunctiva. Iris congested, pupil contracted and does not react to light. Marked photophobia. 10/3/22—Panophthalmitis which later ended in phthisis bulbi.

No. 12. October 11, 1922 O. S.—Injected 0.02 c.c. of pneumococcus culture in the anterior chamber. Thirty minutes later injected 0.2 c.c. of a 1:100 solution of optochin in anterior chamber. 10/13/22 O. S.—Exudate in anterior chamber, pupil contracted. Iris congested. 10/16/22—Considerable circumcorneal congestion. Precipitates on posterior of the cornea. Pupil contracted and does not react to light. 10/20/22—Eye whiter and gradually clearing up. 11/10/22—Eye white and quiet and is normal except for discoloration of the iris at site of the injection of the optochin.

No. 13. September 29, 1922 O. D.—Injected 0.05 c.c. of pneumococcus in the vitreous followed by 0.2 c.c. of a 1:1000 solution of optochin. 9/30/22—Marked chemosis with purulent discharge from conjunctiva. Pupil very small and does not react to light. 10/3/22—Panophthalmitis which resulted in phthisis bulbi.

No. 14. October 11, 1922 O. S.—Injected 0.02 c.c. of pneumococcus culture in anterior chamber. Twenty minutes later injected 0.25 c.c. of 1:100 solution of optochin. 10/12/22—Anterior chamber cloudy, pupil contracted and does not react to light. 10/16/22 O. S.—Exudate in anterior chamber nearly gone, very little circumcorneal congestion, few precipitates on Descemet's membrane. Pupil well dilated and reacts to light. 11/5/22—Eye is normal except for discoloration of the iris at site of injection of optochin.

No. 15. September 29, 1922—Injected 0.05 c.c. of pneumococcus culture in anterior chamber followed 10 minutes later by 0.2 c.c. of 1:100 solution of optochin. 9/30/22—Marked precipitates in anterior chamber. Pupil small and does not react to light. 10/6/22—Aqueous clear, few precipitates on posterior surface of cornea. Pupil fairly well dilated and reacts to light. 11/2/22—Eye normal except an atrophy of iris at site of injection of optochin.

## DISCUSSION.

From the foregoing experiments it would seem that a very small quantity of optochin when injected directly into the vitreous destroys the function of the eyes to such an extent that the pupil will not even react to light. When pneumococci, Type 2, are injected into the vitreous a much greater quantity of ethylhydrocuprein than that which will destroy the pupillary reaction when injected into the vitreous will not inhibit the growth of the pneumococci, consequently optochin has no therapeutic value in infections with Type 2 pneumococcus in the vitreous. On the other hand 0.25 c.c. of a 1:100 solution of optochin may be injected in the anterior chamber without any apparent deleterious effects on its structures except that it causes an atrophy of the anterior layers of the iris with a resulting discoloration at the site of the injection. 0.2 c.c. of a 1:100 solution of ethylhydrocuprein inhibit the growth of Type 2 pneumococcus in the anterior chamber when the drug is injected in this chamber.

The increased reaction of the anterior of the eye in those cases where the pneumococcus was injected as well as the ethylhydrocuprein is explained by the great amount of toxins present in the amount of culture injected. Likewise the precipitates present for 8 to 12 days later on the posterior surface of the cornea were probably clumps of leucocytes in reaction to the presence of the toxins.

From these findings it would seem a perfectly rational therapeutic procedure, when we have a known pneumococcus infection in the anterior chamber to inject 0.2 c.c. of a 1:100 solution of ethylhydrocuprein hydrochlorid into this chamber.

## CONCLUSION.

1. Ethylhydrocuprein when injected into the vitreous in very small quantities destroys the sight of the eye.

2. A quantity of the drug sufficient to destroy the sight when injected into the vitreous will not inhibit the growth of pneumococcus, Type 2, in this body.

3. When 0.20 c.c. of a 1:100 solution of ethylhydrocuprein is injected into the anterior chamber it does not impair the physiologic function of the eye.

4. 0.2 c.c. of a 1:100 solution of the drug inhibits the growth of pneumo-

coccus, Type 2, when injected into the anterior chamber, and can therefore be considered a rational therapeutic measure in that type of infection in the anterior chamber, either following ulcers, or perforating wounds of the cornea, or following operation procedures.

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## ORBITAL ABSCESS IN AN INFANT; EVACUATION THRU A FISTULOUS ORIFICE IN THE BULBAR CONJUNCTIVA.

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A baby 8 weeks old had proptosis, swollen lids, and pus in the conjunctiva. The pus came from an opening in the temporal bulbar conjunctiva, and was pressed out by manipulation. Discharge ceased, radium was used for induration and exophthalmos, which subsequently disappeared. Read before the Section on Ophthalmology of the Philadelphia College of Physicians, February 19, 1925.

In the early part of December last a baby eight weeks old was referred to me with what appeared to be an abscess of the orbit, in itself a rare condition in an infant, the eye in the affected orbit, the left, being proptosed directly forward, the lids swollen and congested, and pus bathing the lashes.

The cornea was clear and the pupil, the same size as that of the fellow unaffected eye, reacted promptly to light stimulus. Upon careful superficial palpation no thickening of the orbital rim or presence of a growth within the orbit could be differentiated; upon firmer pressure, a gush of pus exuded from between the lids, with perceptible lessening of the tension within the orbit and in the degree of the exophthalmos. The pus was washed away and pressure again applied, when it was ascertained that the discharge came from a small fistulous orifice in the temporal bulbar conjunctiva, on a level with the horizontal axis of the the cornea and about 3 or 4 mm. from the limbus. Careful manipulation and pressure was repeated again and again giving exit to a remarkably large quantity of thick pus from this orifice, and greatly diminishing the degree of the exophthalmos. Injunctions were given to the mother to repeat this procedure every few hours, to flush the sac repeatedly with a solution of boric acid, and to instill frequently a 20% solution of argyrol into the conjunctival sac.

I saw the infant daily and at each visit a large quantity of pus was evacuated thru the small orifice in the conjunctiva, the degree of the exophthalmos being still further reduced. After several days, however, it became impossible to express more than a slight amount of pus, and the eyeball could no longer be pushed back into the orbit. A probe was accordingly introduced into the fistulous opening, carried posteriorly well back of the eye and passed in all directions in the hope of opening up some pocket of pus. The result of this exploration, however, was negative.

As there now seemed to be some evidence of induration along the inferior orbital rim, an incision was made at the lower outer angle of the orbit, but no pus escaped. Several more days passing without the escape of pus, and the exophthalmos still persisting, the possibility of a growth in the orbit was thought of and the case was referred to Dr. Pancoast, at the University Hospital, for radium.

Radium application was made and whether in consequence of it, or, what is more likely, by reason of an advance in the inflammatory process, the free evacuation of pus thru the fistula was again established, and continued until the orbit was entirely drained and the eye receded into its normal position.

A few days after the case came under my observation a small shallow ulcer appeared on the cornea, at the limbus adjoining the fistulous orifice. Atropin and cleansing applications were employed, and the ulceration remained localized and disappeared entirely some days after the discharge ceased exuding from the fistula. A recent ophthalmoscopic examination showed no evidence of the optic nerve having been injured by the stretching or the severity of the inflammation to which its infraorbital portion had been subjected.

Altho I can remember the case of a child with orbital abscess which evacuated itself thru the lower conjunctival cul-de-sac, I never saw previously the pointing and draining of such an abscess thru the bulbar conjunctiva, tho such cases are to be found in the literature, usually in connection with the insertion of a rectus muscle.

In the first edition of the Graefe-Saemisch Handbuch, Berlin states "If the pus in cases of orbital abscesses does not evacuate itself spontaneously thru the conjunctiva or lids, it will escape either thru the lamina papyracea of the ethmoid into the nose, thru the floor of the orbit into the antrum, or externally thru the zygomatic fissure, or even thru the superior orbital fissure into the brain."

Orbital abscess in children is a rare condition and the etiology often obscure. In Birch-Hirschfeld's experience the factors operating in such cases may be grouped as follows: (1) direct infection of the retrobulbar orbital tissue in consequence of wounds, operations, and the entrance of foreign bodies; (2) inflammation from neighboring parts; (3) embolic.

Included in the first category are those in which the wounds and agents were of a gross, severe nature, and a smaller class in which the injury to the tissues was extremely slight. Leplat cites an instance where an abscess followed a slight blow from a billet of wood; Sichel, the sting of an insect. In the case I have reported the only etiologic factor seemed to be a slight scratch on the upper lid which the mother noticed shortly after the birth of the child. Labor had been easy and no forceps were applied. The attending physician was unaware of any injury to the eye or lids.

Morax has recently drawn attention to this class of cases and described how staphylococci circulating in the blood may infect the site of a contusion endogenously, and may give rise to osteomyelitis in cases in which there had been no wound in the integument. In my experience the most frequent cause of orbital infection in children has been disease of the superior maxilla, as I have pointed out elsewhere, in a paper before the A. M. A. in 1912 entitled "Orbital Cellulitis from Disease of the Superior Maxillary in Children." In this paper I reviewed the anatomy of this bone and pointed out that it is especially liable to be affected in infancy owing to the cancellated structure of the bone at that age. Infection of the alveolar border by dirty nipples in nursing was held to be a not infrequent cause of disease of the bone.

Orbital abscesses may arise in such cases without the body of the bone being involved in a general osteomyelitic process, by reason of the peculiar anatomic structure of the alveolar process in early life, in consequence of which the pus from the alveoli gains ready access to the orbital cavity by following the channels of connection between the first and second molar teeth and the alveolar canals of the second row of teeth, as well as the channels which unite this latter row with the floor of the orbit.

Disease of the superior maxilla with orbital involvement may also be occasioned by disease of the antrum, as a series of cases attest. F. Krauss has reported before this Section a case of this nature in a four months old child. It is not unlikely that orbital cellulitis following infectious fevers proceed from a primary involvement of this cavity.

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# OCULAR MANIFESTATIONS IN SERUM SICKNESS.

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This paper is based on 75 cases studied in the Contagious Department of the Cincinnati General Hospital. The general symptoms, a rise followed by a fall of blood pressure, fever, edema, rash, joint and muscle pains and prostration, occur eight or ten days after injection. The ocular symptoms are probably due to the vascular changes and proportioned to the severity of the latter. There is hyperemia of the optic disc and in severe cases noticeable swelling.

## GENERAL SYMPTOMS.

The serum sickness encountered after the administration of diphtheria antitoxin is a familiar and extensively studied phenomenon. The lesions and reactions in all species are probably fundamentally produced by the same mechanisms, and differ by reason of the difference in anatomic structure and physiologic reactions. Thus, no one symptom or group can be said to be characteristic in all animals. There is one manifestation, however, that can be considered as an accompaniment in all instances of anaphylaxis, of which serum sickness is a phenomenon, and that is *vascular dilatation with serous exudation.*[1] Schultz[5] noted a primary contraction of all smooth muscles during serum anaphylaxis.

The characteristic initial rise in blood pressure then may be due to a vasoconstriction of the systemic arteries, followed by a paresis and a fall in blood pressure. It is then that the other signs of vasodilatation begin. In man, the lesions are largely disturbances of the vasomotor system. Hyperemia with serous exudation and resulting edema are the most frequent, and, according to the organ or organs involved, contribute most of the symptoms. The symptoms most usual are edema, rash, joint and muscle pains, fever, prostration and adenitis. Since these symptoms are more or less characteristic, it was thought that a study of the ocular phase would be of interest.

The various reactions produced by injections of diphtheria antitoxin, the different degrees of reaction of different individuals to equal amounts, and of the same individual to differently timed injections, are most interesting. The average reaction after intravenously injected antitoxin usually starts in from fifteen minutes to half an hour, with beginning elevation of temperature, and later by a chill. The temperature rises rapidly in the average case to between 102° and 103°, and in some instances to 104° or 105°. The high rises are alternately attended by periods of heat and cold, with usually profuse perspiration.

The above signs are based on the intravenous injection of 10,000 units. The average occurrence of serum sickness is from eight to twelve days after the injection, unless the individual is hypersensitive, in which case the reaction is *immediate*, i. e., in 24 to 48 hours. The reaction is attributed to the continued formation of antibodies even after the serum disappears, so that if a second injection be given some months after the first, an *immediate* reaction occurs within 24 to 48 hours. If the second dose be administered more than a year after the first, the cells may still be "keyed up" to such an extent that an *accelerated* reaction in from 4 to 7 days is produced. If the serum is administered intramuscularly in this case, there may be a local reaction around the site of injection followed by a general reaction in from 4 to 7 days. This is an *immediate*, followed by an *accelerated reaction*. In the average case then, anaphylaxis may be expected if the second injection is given within a year after the first. If sensitivity is suspected, desensitization can be accomplished by an injection of 5 c.c. of horse serum and the rest of the required amount in ¾ of an hour later.

The average case of serum sickness presents a rise in temperature, with a general feeling of discomfort. Arthritic and muscular pains are complained of about the time the urticaria is manifest. The patient may present only a few wheals, or he may be literally covered. The usual symptoms then are rise in temperature, joint and

muscle pains, edema, rash, and occasionally adenitis.

OCULAR SYMPTOMS.

The ocular behavior in seventy-five individuals receiving diphtheria antitoxin intravenously was studied before injection, during the early febrile reaction, the period following, and then during the urticarial stage and the period following.

The examinations were made after instillation of 1% homatropin before injection. Observations were made before administration, every half hour immediately after (during the febrile reaction), then at intervals after the subsidence of the reaction, and again during the urticarial stage and at intervals thereafter. The eyes were likewise examined externally for any variations that might be found. A few cases (ten) were examined without homatropin, in order to observe pupillary reaction.

*External Reaction.* In four of the ten cases untreated with homatropin, the pupils dilated regularly and equally, and were quite sluggish to direct light and accommodation. These pupils were dilated enough to admit of a thoro ophthalmoscopic examination without mydriasis. Kodama[2] noted the dilatation of the pupils in his various methods of administration of horse serum to guinea pigs. He also mentions a vasodilatation of the conjunctival vessels, which was also evident in the recorded series, as follows:

First—Clear during the first 15 to 30 minutes. Second—Slight vasodilatation of conjunctival vessels, with increase in temperature. Third—Permanent, moderate injection or increased injection during the febrile reaction.

There was profuse lacrimation in six of the ten, including the four with dilated pupils.

*Fundi.* The eyegrounds presented the interesting reactions. The general degree may be divided into three classes:

1. Those showing no reaction. 2. Those showing moderate reaction. 3. Those showing intense reaction. The ocular reactions were presumably in direct proportion to the general reaction, viz., severity of the chills, nausea, prostration, general feeling of illness, and the height of the temperature.

Examination of the fundus in the case of average reaction, i. e., in the cases with moderately high temperature (101°-100°) or even those with slightly higher, during the first half hour; no marked change, if any, was manifest. With the increase of the symptoms, however, and the beginning of the periods of heat and chills, with a steadily rising temperature, perhaps one hour to one and one-half hours after injection, a beginning diffuse hyperemia of the disc and retina was usually noticeable. There was a distinct dilatation of the retinal arteries and veins. As the situation progressed, the capillaries of the disc became quite pronounced, and in most cases the hyperemia was at its height in two to three hours. During this time there was a concomitant elevation of the disc cup and the emergence of the vessels seemed less distinct. Even in a moderate case, the surface of the floor of the cup was occasionally flush with the surrounding retina. In cases showing severer reactions, there developed definite papilledema (20%) which was measurable with the ophthalmoscope. Besides the evident edema of the disc, there was also a marked increase in the "watered silk" appearance of the retina, and in severe reactions the appearance was akin to condensing moisture on a glass pane.

As these processes continued, some patients began to notice what they termed "a blurriness" in vision. The outlines of objects lost their distinctness, and complaint of "spots before the eyes" was not uncommon. This symptom was noticed by 10% of the patients upon being questioned. Those who so complained all showed the most marked reactions. The cases came in the third class, showing intense reactions. Unfortunately, these were too ill to have visual fields carefully taken during the height of the reaction. Fields taken afterward proved to be normal.

This entire picture held until a short time after the subjective symptoms of the reaction were over, and then began

to decline to four hours after injection. There was general subsidence of the hyperemia, with slow but steady reappearance of the cup (in those in whom it had disappeared) with a return of the clear disc.

Seven days later an examination was again made to observe any reaction that might occur during the time for inception of the urticarial stage. Fifteen of the seventy-five had urticarial reactions. As the urticaria increased in intensity, it was noted that a similar ocular phenomenon occurred as after injection. Here, as before, the reaction was proportional to the severity of the rash, and subsided in like manner with the decline of the reaction. The administration of adrenalin for the rash had a temporary effect on the ocular manifestations (that of vasoconstriction), but they reappeared after the adrenalin had spent itself.

There were two very intense reactions, one after injection in a colored lad, 12 years, and one during the urticarial stage in a white man, 21 years.

CASE No. 1. C. W., colored, 12 years. Pharyngeal diphtheria. Small patch on postpharyngeal wall. Ill for 1½ days. Temperature on admission 99.5.

1:00 P. M. Ocular examination normal, except for a slight injection of the palpebral conjunctivae. Fundi normal. Received 10,000 units of antitoxin intravenously.

1:30 P. M. Temperature 102.5.

Ophthalmoscopic examination. Retinae—general hyperemia with capillaries distinct. Discs—capillaries quite distinct with elevation of cup. Veins larger in caliber than before, as are arteries. Arteries appear somewhat tortuous.

1:45 P. M. Marked increase in hyperemia, especially of the disc. Cup not seen.

2:00 P. M. Temperature 104°. On questioning, patient says object looks "blurry." Ophthalmoscopic examination practically the same as before.

2:30 P. M. Temperature 104.5. Still complains of "blurriness of vision." Seems quite ill. Patient is having alternate chills and hot spells.

3:00 P. M. Temperature 105°. Retina intensely red. Discs greatly hyperemic. Capillaries distinct. Outlines of discs very indistinct. Evidently a considerable edema of the disc. Height measures 1 D. Patient is quite ill, and does not respond clearly to questioning, as before.

3:30 P. M. Temperature 103°. Patient much more comfortable than ½ hour before. Chills are greatly diminished, and patient is perspiring profusely. Ophthalmoscope. Slight diminution in general hyperemia. Disc outlines still somewhat hazy, but more clearly defined than at 3:00 P. M. Vessels about the same caliber. Arteries are still dilated and tortuous.

4:30 P. M. Patient quite free from subjective symptoms. Appears quite recovered, altho weak. Claims that he sees perfectly well. Ophthalmoscope. General hyperemia greatly diminished, but by no means absent. Disc outlines fairly clear. Cups not visible. Observations thereafter at half hour intervals showed general subsidence.

8:00 P. M. Hyperemia seems quite subsided. Disc outlines clear, but capillaries are still visibly dilated. Cup is distinct. Vessels still dilated, but not as greatly as before. Arteries do not appear as tortuous.

The following morning general hyperemia apparently disappeared. Discs quite normal in outline and color. Vessels of normal caliber. All parts apparently as before injection.

The reaction of the white man, mentioned above, was similar to this one. The primary reaction was, in every way, similar to the above reaction, and then, during a very violent siege of serum sickness eight days later, the patient developed a severe, generalized urticaria. His entire face was livid, his lips were swollen, his chest, back, abdomen, and extremities were completely spotted with wheals, and he suffered severely. The temperature rose from 98.5° to 102.5°, and he complained of a terrible itching.

The ocular manifestations began as before, only this time there was added a violent, diffuse conjunctival injection. The patient, on being questioned, began early to complain of a visual impairment, as before. Ophthalmoscopic

examination, at regular intervals, revealed a rapidly developing haziness of the upper nasal sector of the discs, more marked in the right eye. There was also a concomitant increased capillarity of both discs. The condition held sway for fourteen hours without much relief from repeated administrations of adrenalin. With the subsidence of the urticaria, beginning fourteen hours after the onset, the ocular phase slowly receded. The urticaria disappeared sixteen hours after the onset of the reaction. The ocular phase subsided much more slowly, the fundi appearing quite clear twenty-eight hours after the onset.

In this case, the striking feature was the reappearance in exaggerated form and continuance of the secondary reaction.

COMMENT.

These two cases represent, perhaps, the most severe types of manifestations, but then they appear to be simply magnified duplications of the average reaction that comes with the usual rise in temperature, chills, and distress following intravenously injected antitoxin.

The intramuscular injection of serums entails different conditions at the start. The general reaction is very much less, more delayed in its onset, and more protracted. This is the result of a slow absorption and diffusion, minute quantities being released, over an extended period. In fifteen cases of very early diphtheria, antitoxin was given intramuscularly, and the reactions noted. The average case showed no marked rise in temperature beyond a degree from twelve to twenty hours after injection. The general ocular manifestations were almost negative during this time. In two cases that seemed to be particularly sensitive, there was general distress, feeling of nausea, dizziness, and headache. Both of these cases showed a slight hyperemia of the retinae and discs. The outlines of the discs lost none of their clearness, and there were no complaints of any change in visual acuity. The reactions during the uricarial stage were different. They were quite as marked in the ten cases who developed wheals as those who received the serum intravenously.

ANIMAL EXPERIMENTS.

A dog, weighing 7½ pounds, was placed upon the dog table. The ocular fundi were examined carefully and conditions noted. 5,000 units of stock diphtheria antitoxin (same as used for patients) was injected intravenously.

1:20 P. M. Antitoxin injected. Rectal temperature, 98.4.

1:30 P. M. Ophthalmoscope examination negative.

1:45 P. M. Ophthalmoscope examination negative.

2:00 P. M. Ophthalmoscope examination, retinae, beginning slight dilatation of the vessels. Disc, beginning hyperemia with commencing dilatation of disc capillaries. Temperature 98.8°.

2:30 P. M. Temperature 102°. Chills. Ophthalmoscope examination, retinae definitely hyperemic. Vessels markedly dilated, forming a capillary ring around the edge of the cups. Branches of larger capillaries are clearly visible.

2:45 P. M. Temperature 101°. Animal apparently less restless.

3:15 P. M. Temperature 99°. Animal quiet.

Ophthalmoscope examination, no marked change. There was a gradual subsidence of the hyperemia, with returning of distinct disc outlines, which lasted 5 hours.

Dog No. 2, 6 pounds. 5,000 units intravenously.

Same result, varying only in degree of reaction and time.

Guinea Pig No. 1. 512 gm. Placed on the animal board. Ophthalmoscopic examination made and then 5,000 units of the same type stock antitoxin was injected intraperitoneally. Time, 2:00 P. M.

2:15 P. M.. No change.

2:30 P. M. Slight general hyperemia of fundus and papilla. Also considerable conjunctival injection.

3:00 P. M. Injection quite marked. Chills present.

4:00 P. M. General progressive subsidence in injection of fundus and conjunctiva.

5:00 P. M. No further injection.

Guinea Pig No. 2. Same result as above, with variations as to time and degree.

SUMMARY AND REMARKS.

The study of the ocular manifestations in serum sickness offers an opportunity for studying the reactions in living tissues conveniently and directly, thruout an entire course. The fundus oculi presents for inspection a vascular tissue represented in the choroidal vessels, the ciliary system and capillaries of the retinal vessel system. The disc presents nonmedullated nerve fibers. Thus, we may study at close range fundamental processes, such as vascular associated with nervous manifestations, in vivo.

The vascular manifestations are perhaps the most striking and the most important concerned. In describing the vascular changes in the disc, *hyperemia* is used advisedly, for *increased capillarity* would not adequately describe the reaction. Hyperemia, then must not be construed to indicate beginning inflammation of the disc, but a marked vasodilatation dependent on the serum reaction. It may be noted that with the increase in dilatation of the vessels, come the disc and tissue changes. The disc vessels become prominent first, giving a pinkish tinge to the disc. This being quite early (within the first 15 minutes) precedes a beginning hazing of the disc margin, making it probable that there is an early swelling, due to the vascular dilatation, with exudation into the surrounding tissue. This gives the characteristic onset by clouding the nasal side of the disc, somewhat to the upper border, probably because of the arrangement of blood vessels[3] and nerve fibers on this side.

The direct action of the serum itself, as brought to the tissues, probably does not enter primarily into the ocular reaction. The primary response is a vascular one. The retina exhibits the increased "watered silk" appearance, a condition which may well be a manifestation of the serous exudate and the "blurriness" in vision, being due to increasing edema. The visual fields before, during, and after the reaction have not as yet been investigated. No cases of permanent impairment in vision have been met with in this series, and the only cases of optic neuritis found in the literature are those reported by Mason[4], who observed neuritis after the use of intraspinal sera in two cases, without permanent impairment.

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## TREATMENT OF PHLYCTENULAR INFLAMMATIONS OF THE EYE.

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Phlyctenular disease of the eye has been produced by instillations of tuberculin, staphylococcus vaccin and other substances. In the writer's experiments tyraminum hydrochlorid, and solutions of casein, gave similar results. The suggestion is made that phlyctenules occur in eyes sensitized to foreign proteins, and not as a specific reaction to one substance. Treatment with injections of cow's milk, with proper dosage and interval, are reported to have given good results.

There are many theories on the etiology of the phlyctenular inflammation of the eye, and many reports on pathologic experiments regarding its causation have been published. Rosenhauch, Weekers, Leber, Rubert, Stargardt and others succeeded in producing the phlyctenular inflammation by dropping tuberculin or staphylococcus vaccin into the conjunctival sacs of rabbits

or of guinea pigs, which had been treated with tubercle bacilli or with tuberculin. It can not be regarded as established, that these experiments prove that all phlyctenular inflammations are caused merely by the tubercle bacilli or tuberculin; in the same way we can not think that all cases of conjunctivitis are due to Morax-Axenfeld's bacillus, because we can produce some of them by bringing the bacillus into the human conjunctival sacs. That is why I have tried further experiments on this problem.

I prepared some young rabbits by injecting small doses of staphylococcus vaccin, some with a solution of tyraminum hydrochloricum, or with a solution of casein every day for three weeks. And I was able to produce the phlyctenular inflammation of the eye in some rabbits of every group, as well as in those which had been treated with tuberculin by dropping the same reagents with which they had been treated, or others, into the conjunctival sacs. The details of my experiments were published in the "Klinische Monatsblätter für Augenheilkunde, 71, July-August, p. 141. It is reasonable to say that not only the tubercle bacillus or the tuberculin, but all these reagents can be the cause of this disease. The common element among these reagents is nothing other than "protein," which is foreign to the animals. These foreign proteins cause the general sensibility of the animals, to the same or other proteins, to react with the local specific or nonspecific reactions in their eyes. From this point of view, one can make the fact clear that in many cases of phlyctenular inflammation of the human eye, the scrofulous or exudative diathesis is established, in which condition the remarkable sensibility to proteins can always be proved. Further, it is possible that the eruption of a phlyctenular inflammation is caused by gastrointestinal autointoxication, by resorption of destroyed products of secretions in cases of eczema, etc., and by some bacterial infections when the antigenetic proteins stream into the blood; and, on the other hand, bacteria, or some other substances which directly or indirectly produce the foreign proteins, invade the conjunctival sacs.

If I do not misunderstand the pathology of this disease, in this way, the proteins must be most effectual for the treatment of phlyctenular disease. The effect of tuberculin upon this disease is well known, and still greater efficiency is reported with vistosan, a kind of protein, with staphor, a preparation of staphylococcus, with partigen, etc. Cow's milk is relatively harmless and capable of supplying much of the protein it contains, to improve the resistance of the human body against the specific and nonspecific reactions of various kinds of proteins. It is therefore unquestionably suitable for the treatment of phlyctenular diseases of the eye. Many reports on the effects of the treatment with cow's milk have been published, and I myself have experience of the treatment in some cases.

The injection of cow's milk produces surprising effects in severe cases of phlyctenular diseases, where ordinary treatments have none. Fresh and aseptic cow's milk, which has been boiled for a few minutes, 1 to 4 cc., according to age and general condition of the patient, is injected in the gluteal muscles. The only reaction is a temporary rise in temperature a few hours after the injection, sometimes higher than 104° F., without other unpleasant symptoms. The injections are repeated every three or four days, and the reactive fever decreases in its height each time until it reaches 99° F.

In some rare cases, where this treatment does not take effect quickly, the reactive fever does not decrease as in the former cases, and shows an irregularity of the general temperature curve. This fact can be taken as a proof of the close relationship between the phlyctenular inflammation of the eye and the protein reaction, namely the sensibility of the human organism to protein. In the latter cases one can succeed in the treatment by changing the doses of milk, or the intervals between injections.

# THE BLIND SPOT FOR ACHROMATIC AND CHROMATIC STIMULI.

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BRYN MAWR, PA.

The disagreement in the literature as to a color blind zone at the margin of the physiologic blind spot is here discussed. The apparatus and method used to investigate this point are described. The results of observations made are reported in a series of tables, and the differences in size of the blind spot, for white on black, black on white and colors against white, black and gray backgrounds of the brightness of the color, are shown by charts. Reported from the Department of Psychology, Bryn Mawr College.

The blind spot, discovered by Mariotte[1] in 1668, and often called Mariotte's spot, has been the subject of considerable study and discussion during the last two hundred years. Three problems have arisen in connection with this study: (1) the determination of the limits of the blind spot; (2) the space functions of the region immediately surrounding the blind spot; and (3) the filling in of the blind spot. In this paper the first problem alone will be considered.[2]

Various views, five in all, have been held as to the limits of the blind spot. The first view was proposed by Mariotte who held that the entrance of the optic nerve is insensible to light over its whole extent. This view was immediately opposed by Pequet[3] who believed that the entrance of the retinal artery and vein alone were blind. Volkmann[4] and Coccius were the chief supporters of Pequet, while Hannover[5], Weber[6], Donders[7], Listing[8], Fick and DuBois-Reymond[9], Helmholtz[10], and later Wittich[11] and Aubert[12], upheld Mariotte's view, all affirming that the measurements of the blind spot corresponded more closely with the diameter of the entrance of the optic nerve than with the diameter of the retinal artery at its place of entry, as was contended by Volkmann.

Donders and Helmholtz demonstrated directly that the nerve fibers at their entrance were insensible to light. With the aid of an ophthalmoscope, they projected the image of a flame into the eye so that it fell upon the nerve fibers as well as on the blood vessels. They found that no sensation was aroused in either case.

Weber and Hannover advanced the argument that the defective area could not be limited to the blood vessels, as this would give an irregular and branchlike form to the blind spot rather than an area roughly oval in shape, as is demonstrated to be the case.

The third view, that not only is the entrance of the optic nerve blind, but also the area immediately surrounding it, was advanced by Griffin[13] who found that the diameter of the blind spot varied, within limits, inversely as the intensity of the stimulus. This view was supported by Weber[6], Bjerrum[14], Meisling[15], and Groenouw[16], all of whom found the area of absolute blindness to be surrounded by a zone of relative blindness in which large stimuli are visible but smaller ones are not.

The fourth view has been advanced and supported at different times by Johansson[17], Ovio[18], Polimanti[19], Haycroft[20], and Van der Hoeve. They maintain that surrounding the area blind to colorless stimuli is a narrow zone relatively blind to color. Polimanti, for example, found blue and yellow to have narrower limits than red and green, while Haycroft, using as stimuli painted nail heads on a black surrounding field, found the order from widest to narrowest to be green, red, and blue. Van der Hoeve says the absolute scotoma or blind area is surrounded by a zone of one-eighth to one-fourth degree relatively blind to white, and beyond this is a zone of an average breadth of one-half degree, which is relatively blind to color.

The fifth view was advanced by Ramsay and Sutherland[21]. These writers claim, that beyond the zone of relative or transitional amblyopia to white is a zone absolutely blind to color, varying in width for the different colors. This zone is widest in order for green, red, yellow, and blue. Thus, according to Ramsay and Sutherland, the scotoma is larger for colored than for colorless stimuli and is absolute.

Berens[22], one of the most recent writers on this subject says, "Most authors of today agree that there is a relative scotoma for white and colors surrounding the absolute blind scotoma * * * * Observations of Van der Hoeve and Ramsay and Sutherland are fairly well borne out by the investigations on normal subjects, except that with accurate observers, when using a black screen, daylight illumination, and large colored test objects, the fields for colors, white, and motion all coincide so closely that it is difficult to say that they were not all observed practically at the same time." He states further that the results are erratic and difficult to reproduce, when the determinations are made on a white or light gray field, but that with a black or dark gray screen and constant artificial daylight illumination, his subjects were quite accurate in reproducing their results.

The preceding survey of the literature shows that there is a great deal of disagreement in the results obtained in the work that has been done on the blind spot. Some of this may be due to the individual differences in the eyes investigated, but doubtless the major portion of it may be attributed to the lack of a standard set of conditions under which to work. Until the variable factors, which influence the size and shape of the blind spot, are discovered and controlled there is little chance of doing work on the blind spot with a sufficient degree of precision, or reproducibility, to make it of any considerable value either for practical or scientific purposes. It has been the purpose of the present study to map the blind spots for chromatic and achromatic stimuli, under standard conditions, selected with special reference to the control of the variable factors which influence the results of blind spot determinations. In a later paper the effect of these factors on the size and shape of the blind spot will be shown.

## APPARATUS AND METHOD.

The experimental work was done on the Ferree-Rand perimeter. This perimeter was designed for the control of all of the variable factors which effect the determination of the color fields for a given eye. For a detailed description of the instrument the reader is referred to the following publications by Ferree and Rand: "A New Laboratory and Clinic Perimeter," Journ. Exp. Psych., 1922, vol. v. pp 46-67; "An Illuminated Perimeter with Campimeter Features," Transactions of the American Ophthalmological Society, v. XVIII, 1920, pp. 164-172; The American Journal of Ophthalmology, 1922, v. 5, pp. 455-465; Transactions of an International Congress of Ophthalmology, 1922, pp. 388-423.

Among the special features and devices of this perimeter the following may be mentioned: A means of illuminating the perimeter arc, such that every point on the arc in any meridan in which it may be placed receives light of equal intensity and of daylight quality; a means of controlling the brightness of the preexposure and surrounding field; devices and adjustments for the accurate location of the eye at the center of the perimetric system and for securing a steady and precise control of fixation; special fixation controls for eyes suffering from presbyopia, high degrees of hyperopia and myopia, low central acuity, and central scotomata; attachments for studying the blind spot and for refracting the peripheral field; a tangent screen or campimeter attachment, which can be equally illuminated at every point on its surface and with which all of the fixation devices noted above can be used; etc.

The special features of this instrument which are of particular value for blind spot study are: The provisions for the control of intensity, color and evenness of illumination; the devices and adjustments for the accurate location of the eye at the center of the perimetric system and for securing a steady and precise control of fixation; and the special attachments for blind spot work, providing for the control of the brightness of the surrounding field and other variable factors.

One of these attachments consists of a special carrier in which may be in-

serted as desired the specially prepared paper screens on which the blind spot is to be mapped. These screens are larger than those used for field-taking and extend farther below than above the center of the perimeter arc to accord with the location of the blind spot with reference to the horizontal meridian. These screens may be white, black, grays of the brightness of the color, etc., as the needs of the work may require. On each screen is stamped 24 equally spaced lines or meridians, radiating from the center outwards. These meridians serve as guides for the movement of the stimulus in determining the limits of the blind spot, or when checking up the location of the limit by a second or third determination. After the determination these screens may be removed and filed as a permanent record, from which the outline of the blind spot may be transcribed.

In using this attachment for mapping the blind spot, the perimeter arc is turned into the horizontal meridian and the carrier moved along the arc until its center coincides with the center of the blind spot. The center of the blind spot is located for a given eye by determining the nasal and temporal limits for an achromatic stimulus, white on black, or black on white, and finding the point midway between these limits, the degree of eccentricity of which can be read on the perimeter arc. In centering the carrier the pointer on its back is moved to this degree value on the perimeter arc.

In determining the limit of the blind spot in any meridian the stimulus is passed along that meridian towards the insensitive area until the observer reports its disappearance. Its direction is then reversed until it reappears. The points of disappearance and reappearance should coincide, for an accurate observer. In case of the present study, when they were not found to coincide the average value was taken after several observations, provided that the distance between the points was less than the breadth of the stimulus, 25 min. or 0.4 deg. If the error did not fall within these limits the result was discarded as unreliable. It may be noted in this connection that it was necessary to take the average of the points of disappearance and reappearance in case of only a very few observers. After a little practice the percentage of error of most of the observers was very low indeed.

Frequent rest periods were allowed to guard the eye against fatigue. This is important in blind spot work for color. Some observers fatigue to color so rapidly on the margins of the blind spot that it is advisable to rest the eye for a few seconds between each excursion of the stimulus to or from the blind area. If this is not done the results are erratic and unnecessary changes in hue are obtained. The precaution is not nearly so important when achromatic stimuli are used.

The stimuli employed were small discs 2.5 mm. in diameter, cut by means of a special punch. As already noted these stimuli subtended a visual angle of 25 min. at the eye 33 cm. distant. The chromatic stimuli were obtained from the Heidelberg papers, red, blue, and green; the achromatic stimuli from the Hering white and black papers. For convenience of use these stimuli were mounted at the ends of slender rods, painted to match the screens on which the determinations were made. The perimeter and table on which it stood were painted mid-gray to avoid extremes of brightness preexposure, during the rest periods and the preliminary period of adaptation.

White, black and grays of the brightness of the color, as seen on the edges of the blind spot, were used as screens on which to make determinations. These were taken from the Hering series of standard papers. The gray used for the red and blue stimuli was number 11 of this series, coefficient of reflection 24 per cent; and the gray for the green stimulus was number 6, coefficient of reflection 42 per cent.

The intensity of illumination of the perimeter arc and blind spot screen was kept constant at 7 foot-candles. Constancy of illumination was secured by ammeter and rheostat control of the

TABLE I.

Blind Spots for Achromatic and Chromatic Stimuli: Area in square centimeters.

| Stimulus Field | White Black | Black White | Red Gray | Red Black | Red White | Blue Gray | Blue Black | Blue White | Green Gray | Green Black | Green White |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Case 1..... | 11.2 | 11.6 | 13.2 | 13.8 | 13.5 | 12.1 | 12.6 | 20.0 | 11.9 | 14.6 | 25.0 |
| Case 2..... | 11.2 | 13.0 | 16.2 | 15.5 | 22.5 | 14.0 | 14.1 | 20.7 | 14.6 | 16.2 | 25.0 |
| Case 3..... | 6.0 | 7.8 | 10.9 | 13.3 | 14.6 | 12.0 | 11.0 | 19.8 | 9.9 | 27.3 | 20.7 |
| Case 4..... | 5.7 | 9.7 | 16.2 | 20.2 | 19.0 | 13.3 | 17.6 | 23.5 | 12.2 | 16.3 | 17.5 |
| Case 5..... | 7.1 | 6.7 | 7.1 | 10.5 | 11.9 | 10.1 | 9.9 | 13.5 | 8.0 | 10.6 | 16.2 |
| Case 6..... | 7.2 | 7.0 | 7.3 | 9.9 | 8.7 | 8.7 | 11.3 | 9.3 | 8.7 | 8.4 | 12.7 |
| Case 7..... | 10.0 | 7.4 | 15.2 | 11.4 | 12.6 | 14.5 | 11.6 | 23.8 | 10.2 | 13.1 | 30.8 |
| Case 8..... | 11.6 | 10.0 | 11.0 | 13.0 | 15.2 | 11.8 | 13.0 | 14.2 | 14.3 | 15.8 | 16.3 |
| Case 9..... | 7.2 | 9.0 | 12.3 | 13.5 | 16.9 | 13.0 | 12.4 | 16.3 | 11.6 | 13.0 | 17.9 |
| Case 10..... | 7.3 | 8.4 | 9.6 | 10.4 | 10.3 | 8.9 | 10.2 | 17.1 | 11.4 | 9.3 | 16.7 |
| Average*.... | 8.43 | 9.06 | 11.90 | 13.15 | 14.52 | 11.84 | 12.37 | 17.82 | 12.22 | 14.45 | 19.56 |
| Median..... | 7.85 | 8.70 | 11.65 | 13.15 | 14.05 | 12.05 | 12.00 | 18.45 | 11.75 | 13.85 | 17.70 |

*By average in this and the following tables is meant the arithmetic mean, obtained by dividing the sum of the values by the number of cases. Because of the small number of cases the median value is also given.

TABLE II.

Blind Spots for Achromatic and Chromatic Stimuli: A Comparison of the Size, for Color and for White on Black.

| Field | Average Area in sq. cm. Gray | Black | White | Per cent larger for color than for white on black Gray | Black | White |
|---|---|---|---|---|---|---|
| White stimulus ..... | .... | 8.43 | .... | .... | .... | .... |
| Red stimulus ..... | 11.90 | 13.15 | 14.52 | 41.1 | 56.1 | 72.1 |
| Blue stimulus ..... | 11.84 | 12.37 | 17.82 | 40.4 | 46.7 | 111.3 |
| Green stimulus ..... | 12.28 | 14.45 | 19.56 | 45.6 | 71.4 | 132.0 |

TABLE III.

Blind Spots for Achromatic and Chromatic Stimuli: Average Diameter in Horizontal and Vertical Meridians.

| Meridian Field | Horizontal Gray Cm. | Horizontal Gray Degrees | Horizontal Black Cm. | Horizontal Black Degrees | Horizontal White Cm. | Horizontal White Degrees |
|---|---|---|---|---|---|---|
| White Stimulus ..... | ..... | ..... | 2.58 | 4° 28′ 36″ | .... | ..... |
| Black Stimulus ..... | ..... | ..... | .... | ..... | 2.58 | 4° 28′ 36″ |
| Red Stimulus ..... | 2.91 | 5° 2′ 36″ | 3.27 | 5° 40′ 18° | 3.48 | 6° 2′ 38° |
| Blue Stimulus ..... | 2.81 | 4° 51′ 30″ | 3.19 | 5° 32′ 14″ | 3.63 | 6° 18′ |
| Green Stimulus ..... | 3.21 | 5° 34′ 14″ | 3.43 | 5° 57′ 8″ | 4.12 | 7° 8′ 50″ |
| | | | Vertical | | | |
| White Stimulus ..... | ..... | ..... | 3.89 | 6° 45′ 36″ | .... | ..... |
| Black Stimulus ..... | ..... | ..... | .... | ..... | 4.16 | 7° 14′ 52″ |
| Red Stimulus ..... | 4.80 | 8° 25′ 34″ | 5.26 | 9° 6′ 38″ | 5.26 | 9° 6′ 38″ |
| Blue Stimulus ..... | 4.76 | 8° 4′ 48″ | 5.05 | 8° 44′ 46″ | 5.50 | 9° 31′ 34″ |
| Green Stimulus ..... | 4.78 | 8° 16′ 50″ | 5.37 | 9° 18′ 22″ | 6.07 | 10° 30′ 28″ |

current operating the lamp used to illuminate the arc of the perimeter. At the beginning of each working period the sensitivity of the eye of the observer was standardized by a twenty minute period of adaptation to the illumination used.

It was impossible in most instances to obtain at a single sitting a complete set of maps between which comparisons were wanted. To guard against comparison being made between results obtained with the eye in a different state of sensitivity the work done at a previous period was always adequately rechecked, the criterion being that the results obtained at different periods must fall within the same limits of error as those obtained at a single sitting.

RESULTS.

A detailed statement of results is contained in Tables I-III and a graphic

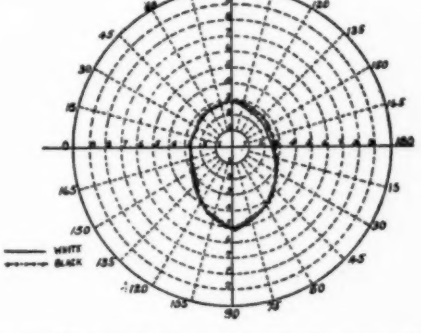


Fig. 1.—Blind spot for a white stimulus on a black field and a black stimulus on a white field (10 observers). In these charts half the actual at a distance of 33 cm. is represented.

representation in the accompanying maps, Figs. 1-5.

The areas of the blind spots for the different stimuli and conditions employed are given in Table I. These areas were measured by means of a planimeter. They represent the size of the blind spot at a distance of 33 cm. The advantages of measuring the area for comparative purposes is obvious. Only a rough knowledge of comparative sizes can be had from the inspection of the plotted graph or map of this area.

This statement applies to the treatment of the results for all types of field study. If a comparison is to be made, with the minuteness and precision needed both for laboratory and diagnostic purposes, a single value or index must be had; which can be taken as fairly representative of the retinal areas under investigation. In case of the blind spot no more accurate and convenient index of the actual extent of the insensitive area can be found, perhaps, than the areas of the plotted graph, provided that the limits of sensitivity have been determined and plotted in a sufficient number of meridians. The measurement of this area by means of a planimeter is extremely quick and simple.

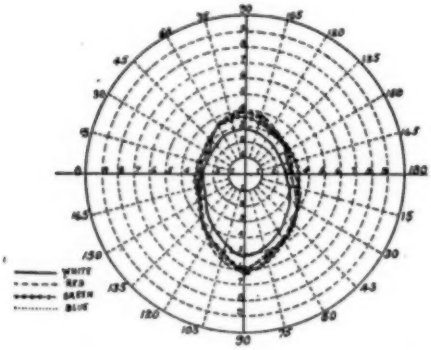

Fig. 2.—Blind spot for red, green and blue stimuli, on gray of the brightness of the color; and for a white stimulus on a black field (10 observers).

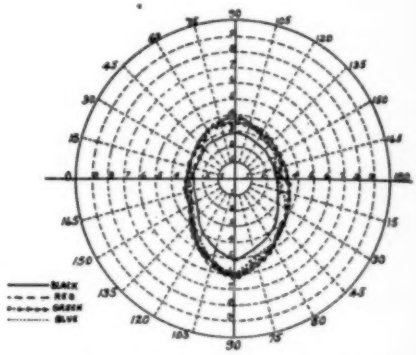

Fig. 3.—Blind spot for red, green and blue stimuli on gray, of the brightness of the color; and for a black stimulus on a white field (10 observers).

In Table II is shown the difference in size of the blind spots for the different stimuli and conditions employed. This difference is expressed both in square centimeters and per cent. In computing the values shown in the table the area of the blind spot for white on black was used as the base. This relation of stimulus and background was chosen because of the frequency of its use in clinical work.

The results contained in these tables may be summarized as follows:

(1) The blind spot for achromatic stimuli averages 7.6 per cent larger on

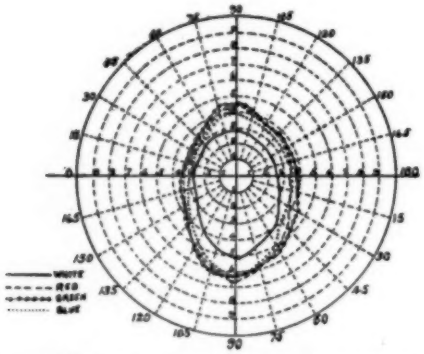

Fig. 4.—Blind spot for red, green, blue and white stimuli on a black field (10 observers).

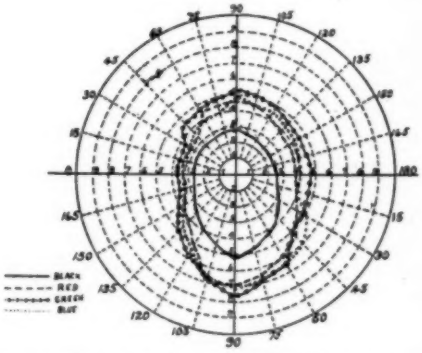

Fig. 5.—Blind spot for red, green, blue and black stimuli on a white field (10 observers).

a white than on a black field. That is, a black stimulus on a white field gave a slightly larger blind spot than a white stimulus on a black field. This difference is in the direction that might be expected. That is, the influence of the irradiation of the white excitation on the retina would tend to increase the area stimulated in case of the white stimulus on the black field, and to decrease it in case of the black stimulus on the white field. Probably because of this, too, a white stimulus on a black field always appears larger than a black stimulus on a white field.

(2) The blind spots for the chromatic stimuli were all much larger than for the achromatic stimuli. The difference is so great as to leave no doubt as to the conclusion that should be drawn. On the gray of the brightness of the color they were 40-45 per cent larger than those for the achromatic stimuli; on the black field, 47-71 per cent larger; and on the white field, 72-132 per cent larger.

(3) Not only the size but the order of ranking as to size varied with the surrounding field. From largest to smallest the order for the white field was green, blue, and red; for the gray of the brightness of the color and the black field the order was green, red, and blue.

In Table III is given the average extent of the blind spot in the horizontal and vertical meridians. These dimensions are given both in centimeters and in visual angle subtended at the eye. The average distance of the center of the blind spot from the center of the field of vision for the achromatic and chromatic stimulus was 16.5°, the range being from 14.5-17.5 degrees.

In a series of papers to be published later, not only the factors affecting the results of blind spot determinations will be discussed, but a diagnostic scale will be prepared for use in clinic work.

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# OCULAR COMPLICATIONS OF CHOREA.

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The conceptions of chorea now held by neurologists are briefly reviewed. Among the more important ocular complications described in the literature are mentioned; obstruction of the central retinal artery or its branches, sometimes followed by optic atrophy, optic neuritis, disease of the oculomotor nuclei and resultant weakness or palsies. Three cases, coming under the latter heading, are reported. Read before the Ophthalmological Section of the Baltimore City Medical Society, January 22, 1925.

While this society is not primarily interested in the neurologic aspect of the subject which is here presented, it seems impossible to do justice to it unless some of the neurologic phases of it are considered, with especial reference to etiology, diagnosis and morbid changes.

So little is known concerning the etiology of true chorea that a few words will serve to cover it. That it is an infection seems undoubted, its onset, its most frequent occurrence in the spring of the year, its complications both cardiac and arthritic, its course, the recurrences which it shows, all seem to point to some organism as its starting agent. Chorea is not a disease which is often fatal; and as a result material for pathologic study is not often obtained. Such cases as have been studied show cerebral changes mostly on the convex surface and in the meninges covering it.

C. L. Dana reports the case of a man who died during a recurrent attack of chorea, the original onset having been twenty years before. He had had many attacks, the movements being so severe that he would be confined to bed for weeks at a time. Postmortem examination of the brain showed a chronic proliferative meningitis of the covering of the convex surface including the motor areas, with some involvement of the underlying cephalic structure. Bacteriologic studies isolated an organism resembling the diplococcus lanceolatus.

This case of course was one of unusual duration and severity, and it does not seem probable that such marked changes would be found in an ordinary case of chorea. It seems possible that the movements are due to an excitation of the motor cortex by an invading organism producing a meningoencephalitis which is capable of a return to normal but may continue and result in intermittent attacks or even a continuous condition.

Dana also reports a series of nineteen cases which were necropsied and in sixteen of them were found cerebral hyperemia, periarterial exudates and hemorrhages, spots of softening and occasional emboli, extending deep into the motor tracts as far as the lenticular nuclei and the thalami. It is of interest that practically the same findings were recorded by W. H. Dickinson as early as 1876. There were no characteristic lesions of the medulla and the cord. In the ventricles of many was found the staphylococcus pyogenes aureus.

Dana believes that chorea is an infectious disease but that certain factors act as underlying causes as do Osler and McCarthy, namely brain and eyestrain, a tendency in certain families to rheumatism, in the spring months.

The most noticeable thing about a case of chorea is the movements, and usually but little difficulty is found in making the diagnosis. The condition most likely to be misleading is the so-called habit spasm or tic, as it was named by the Salpetriere school; which describes it as usually beginning in childhood, involving the face, neck and possibly the arms, but seldom the trunk or legs, and differs from true chorea in the rapidity and system of the motions.

A second class of tic occurs with the above symptoms and in addition psychic phenomena accompanied by outcries, obscene or profane words, or a rapid repetition of words, the socalled echolalia.

The motions of chorea are less systematized, less rapid and not so apparently purposive as are those of habit spasm.

Weir Mitchell classified the motions of true chorea as follows:

(a) Cases in which the motions stop on purposive movements, for instance in holding a cup of water.

(b) Those in which such purposive motions increase the chorea movements.

(c) Where the chorea is evident only on making some voluntary motion.

(d) Where there is an alteration in the type of the involuntary motion when some voluntary act is performed.

He regards chorea as a definite irritation of the motor areas.

Eshner regards chorea as the result of an infection, and cites the joint and heart complications as a proof of this. There is no rise of temperature. The motions of true chorea are purposeless, beyond control, jerky and incoordinate —but cease during sleep. At times mental symptoms are marked. He feels it does not result from eyestrain.

Concerning this last point de Schweinitz made an investigation, examining the eyes of fifty choreic children. The findings of interest were that there were no fundus lesions typical of chorea observed, but all of the children were hyperopic and the changes which frequently accompany this condition were noted. He found no cases where ocular strain seemed to be the cause of the motions. The writer may say concerning this point, that while he believes chorea is an infectious disease he has seen many cases where the correction of the error of refraction has apparently influenced the course of the disease; and he believes that all choreics should have a most careful and thoro ocular examination, and correction of such error of refraction as may seem sufficient to justify it.

Organic ocular changes occurring during attacks of chorea are rare but may be severe and cause serious disability. One such is embolism of the central artery of the retina, eleven cases of which have been reported, nine quoted by Leber[1] in volume seven of the Graefe-Samisch Handbook, one by Arnold Knapp[2] in the Archives of Ophthalmology in 1918, and one by the writer[3] at the Section of Ophthalmology of the College of Physicians of Philadelphia, in 1924.

Of the cases collected by Leber the following seem worthy of extended comment.

Swanzy[4] in the Royal London Ophthalmic Hospital Reports for September, 1875, reports a "Case of Sudden Amaurosis, associated with Chorea." It was a girl ten years of age, who was seen by him two weeks after the loss of vision of the left eye, when he first saw it the eye being totally blind. The artery of the retina was small, pale but not quite empty—veins also being smaller than normal—the retina was hazy around the disc, with a broad zone also in the temporal portion, surrounding a crimson spot at the macula.

The child had chorea, the left arm and leg being especially involved—this having made its appearance almost simultaneously with the blindness. The health of the patient had been previously good, except for mild attacks of whooping cough and measles. No cardiac disease could be found. Both the chorea and the vision improved and in three weeks after her first examination, she could count fingers at five inches. The disc gradually paled, and the inferior nasal branch of the artery collapsed, later to have its blood column return.

Swanzy believed this was a case of either embolism of the central artery or hemorrhage into the sheath of the nerve, most probably the former. Gowers[5] in his "Medical Ophthalmoscopy," accepts this as a case of embolism, but makes the peculiar mistake of saying, "the condition of the heart is not mentioned," whereas Swanzy plainly states, "no cardiac disease could be found."

Gowers mentions also another case recorded by Foster, a child seen by him some time after the loss of the sight of one eye, following a prolonged attack of chorea. The disc was atrophic and the retinal arteries very small.

W. G. Sym[6] reported in 1888, "A case of optic atrophy following chorea." His patient was a boy seventeen years

of age who, ten years before he was seen by Sym, had suddenly lost the vision of his right eye. He was in the Royal Infirmary on account of an attack of chorea. At the time of the visual loss he was walking quietly down the ward, making no exertion and was subjected to no blow or injury. The eye had remained blind as it was when Sym saw him, the vision of the other eye being normal. The disc was atrophic, the retinal arteries very small, but no other changes were present. He had the signs of mitral stenosis. Sym believed this ocular condition the result of embolism of the central artery, and says he was informed by Argyll Robertson that he had seen a similar case, which he had not reported, in a young lady with chorea.

Arnold Knapp[2] in 1918 reported the case of a girl eight years of age, who had had repeated attacks of tonsillitis and later mumps; followed in a month by an attack of chorea involving the left hand, the right foot and the face; followed the next day by the loss of the vision of the left eye. Examination showed the right eye normal, the left being blind with a fixed pupil, normal retinal vessels, except the inferior temporal artery which had collapsed, haze of the retina and a "cherry red" spot at the macula. The heart showed the presence of endocarditis. Knapp believes that the appearance of embolism in this case speaks for the theory which holds chorea to be of embolic origin.

Does it not seem more likely that the infection which causes the endocarditis causes also the encephalitis producing the chorea, and the emboli are sequelae of the heart condition?

The case which the writer showed was seen by him thru the kindness of Dr. Frederic H. Leavitt. A girl, aged 11, developed chorea in August, 1923, and endocarditis in November, 1923. In January, 1924, the right eye became blind. Examination of the ocular condition showed that the left eye had normal vision and was healthy in all respects. The vision of the right eye was found to be faint light perception, no pupillary reaction except consensually, clear media, ocular rotations well performed, disc normal in color, what appeared to be a small plug at the bifurcation of the superior and inferior arteries, the arteries themselves being small, the veins about normal in size and a cloud in the temporal portion of the retina, with the macula spot showing dark red. There were no hemorrhages, exudate or other fundus changes, the appearance was that of a typical block of the central artery of the retina. Later the arteries almost completely collapsed, the inferior one being a very thin line and the disc became atrophic.

Blockade of the central artery of the retina may be from one of three causes, embolus, thrombus or spasm, the last in all probability only becoming permanent where a thrombus is formed. Undoubtedly embolus of the central artery of the retina is a very rare occurrence. Mr. George Coats after examination of a large amount of pathologic material, which had been reported as embolic in origin, found only five cases which he felt were substantiated by microscopic examination. Harms, in order that a diagnosis of embolism be considered plausible where the central artery of the retina is closed, demands that the arteries be normal and there be some diseased area which might be the origin of the embolus. In this case we have both, the endocarditis as a starting point of the embolus and the child's vessels were practically normal. Rare as embolism of the central artery of the retina is, it is more rare as a complication of chorea and when we consider the large number of children with chorea, who have endocardial involvement, it seems we might expect this to happen more frequently.

In speaking of the fundus changes occurring in cases of chorea, Gowers comments on the fact that most of them are the results of hyperopia and not of the chorea, but says he had observed two undoubted cases of optic neuritis which occurred during attacks of chorea, without other ascertainable cause, and which disappeared with the improvement and recovery from the nervous condition. He gives no

other information concerning the patients, apparently attributing the inflammation to the toxin of the chorea and not to some intermediary complication.

Infection severe enough to cause such conditions might be expected to bring about changes in either neuclei or the nerves supplying other ocular structures, but no such seem to have been reported. In three cases at the Infirmary for Nervous Diseases, the writer has seen diplopia appear during attacks of chorea and disappear as the condition abated, physical examination revealing no other cause for the palsy.

The first was J. K., a boy, aged 9, a patient of Dr. Morris J. Lewis, seen first February 13, 1907, the chorea having been present two months. He had homonymous diplopia over the right field and loss of outward rotation of the right eye. There was about 4 D. of hyperopia, with normal corrected vision, but distinct hyperemia of both discs. He made a complete recovery from the muscle palsy.

On July 26, 1907, W. S., a boy, aged 4, was seen on the service of Dr. J. K. Mitchell at the Infirmary. He suffered with chorea which had lasted from the previous December. He had suddenly complained of diplopia, had turned his head to the left and the mother had noticed convergence of the left eye, which, examination showed, could not be rotated outward more than fifteen degrees beyond the line of central fixation; there was a diplopia typical of left external rectus weakness. Except for the chorea he showed no likely etiologic condition, and in three weeks, or by August 19, 1907, he had completely recovered.

The third case of extraocular muscle weakness was A. H., a girl of thirteen, who was admitted to the ward of the infirmary on the service of Dr. Lewis on June 3, 1908, with a very severe attack of chorea involving the whole left side; she also had a systolic murmur and arthritis. On June 17, just two weeks after admission, she complained of diplopia which was due to loss of activity of the right superior rectus muscle. This completely disappeared in ten days, altho the other conditions did not improve enough for her to leave the hospital until two months later.

These palsies may result from involvement of either nuclei or nerves and with the changes which are known to recur in the brain in chorea. The surprising thing is their infrequency, tho, as in all medical cases, a routine study of the eyes in chorea might show that they occur more often than we suppose.

The ophthalmologist is not apt to see cases of chorea until after the neurologist. But it is not rare for him to be the first to be consulted concerning the habit spasm involving the face and lids; and therefore it would seem that he should be in a position to make, at least a tentative diagnosis of the nervous condition and, should it be habit spasm, to make sure that no ocular strain is the inciting factor. Such habits may become fixed unless handled promptly and efficiently.

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# SIDEROSIS BULBI.

S. B. MUNCASTER, M.D.

WASHINGTON, D. C.

In the case here reported, the presence of steel in the eye was not recognized and the eye deteriorated until removal was advised. The X-ray showed a foreign body in the vitreous; and after magnet extraction the eye was improving. Read before the Society of Ophthalmology and Oto-Laryngology of the District of Columbia, January 16, 1925.

Fuchs in his Text Book, defines siderosis bulbi as "the impregnation of the tissue with iron."

The American Encyclopedia of Ophthalmology, under siderosis bulbi, states, "when particles of iron remain for a long period within the eye, or are imbedded in its tissues, all parts of it may be stained a rusty-brown by the deposit of ferruginous salts."

Von Hippel distinguishes two kinds of siderosis: "Exogenous siderosis, due to the presence of a foreign body which is of iron or steel; and hematogenous siderosis, following hemorrhages and due to the iron which is usually present in the blood."

Bunge, in a paper, seems to have been the first to use the term siderosis bulbi, giving as his view that it was due to the dissemination of iron in the eye.

Leber, in 1881, believes that "the iron was dissolved by the carbon dioxid of the tissues and converted into the carbonat. This salt, circulating in the vessels, was finally deposited as an insoluble compound by the acid salts of the blood."

De Schweinitz reports a case in which the spots on the capsule of the lens and discoloration of the iris disappeared three and one-half months after the removal of the foreign body.

J. Gray Clegg states: That siderosis is a rare condition, may be gathered from the fact that during 15 years ending October, 1913, 458,496 new patients were admitted at the Royal Eye Hospital, Manchester, England; of these 111,371 were accidents, and yet one can find only 13 cases which were diagnosed to be siderosis.

Graefe was the first to call attention to the discoloration of the tissues of the eye from retained metallic particles.

From reading on the subject of siderosis bulbi it is gleaned that the most important diagnostic points to note are the change of the normal color of the iris to a rusty-brown, with yellowish dots in the form of a wreath. When the pupil is dilated one will find yellowish-brown spots under the capsule ½ mm. in diameter and 1 or 2 mm. apart, all around the capsule. Sometimes these spots are in the anterior part of the capsule.

When a piece of iron or steel enters the ball thru the sclera into the choroid, or retina, or uvea, an inflammatory condition takes place and uveitis is apt to follow, and an absorption of the iron takes place resulting in a dark brown color in various membranes. The color of a blue iris will change to a deep brown. The lens of an eye will give very little trouble from a piece of iron, except to change color, becoming cloudy.

I have seen a piece of steel that had been in a cornea 21 years, and only a slight brown color appears around the metal.

Last month I had a patient with a phthisis bulbi, the iris had all the appearance of a siderosis bulbi, and the only way I can account for the rust brown color and yellowish dots in the iris, is a probable hemorrhage and iron in the blood. X-ray failed to show any metal.

Mr. C. called at my office for treatment of his eyes with the following history: In December, 1923, he was working in a blacksmith shop in Petersburg, Virginia. As he struck a piece of hot iron with a hammer, he felt something hit his right eye. He went to an oculist who prescribed some "drops;" in a few days the eye felt all right and remained so for five weeks, then vision began to fail. Mr. C. returned to the specialist who thereupon gave him a blood test and treated him otherwise. After finding that the blood and general health were

good he recommended him to Dr. John Dunn, Richmond, Va., for consultation. The two specialists treated him for about four months and finally concluded the eyeball should be removed The patient's cousin in this city advised him to consult me before anything was done. On examination I found sclieritis, iritis with irregular and dilated pupil, and tension below normal. With the ophthalmoscope I found a diseased

Fig. 1.—Case of siderosis; extraction of metal; retention of eyeball with good vision.

condition of the tissues in the lower section of the eyeball with choroiditis, retinitis and patches of pigment, vitreous cloudy, optic nerve hazy, and floating opacities in the vitreous.

With such a condition of the eye I was inclined to agree with the other two specialists, but thinking this trouble might have been induced from a foreign body in the eye, especially after calling to mind that he had felt something hit him in the eye when he struck the hot iron with his hammer, I recommended him to Dr. Groover for an X-ray examination; and fortunately Dr. Groover located the metal in the back of the eyeball. I then had the patient go over to the Episcopal Eye, Ear and Throat Hospital and there I applied the large magnet, but without any result.

I then cut thru the sclera about ¼ of an inch into the eyeball and applied the end of a small magnet, twice without any result. The third time I went far into the vitreous with the curved end of the magnet and on its removal my efforts were rewarded with a sight of the piece of iron adhering to the magnet. The reason the iron did not come out when I first applied the magnet was because it had become encapsulated. I put four stitches in the conjunctiva and removed them in four days. The sight gradually improved from almost nothing to 20/40 and is still improving. The iris that was blue is brown with a yellowish tinge near the margin of the pupil. The pupil is almost normal at the present time. The treatment was a drop of 3% atropin sulphat at my office, and a solution of 1% in his eye twice daily, also hot applications, dark glasses, and so forth.

When the patient was about to return to his home I requested him to see the two specialists and show them the piece of iron that had been removed.

With permission of Dr. John Dunn I quote his letter of October 27, 1924, relative to the case:

"I did not mean you to infer from my letter that I had never seen a case of siderosis bulbi, for as recently as August 20th, 1924, I have full notes of a case with X-ray diagrams showing location of the foreign body, etc. I have, however, never seen a case presenting a similar history, with similar symptoms to that of Mr. C. The patient had been under treatment by another oculist for about five weeks when I first saw him, during which time various blood and other tests had been made; and he had been subjected, in spite of a negative Wassermann, to iodides and mercury. He gave me the history of an apparently causeless failure of vision about five weeks prior to his first visit. Two or three days after his sight began to fail his friends called his attention to the fact that one pupil was larger than its fellow. This had gradually enlarged until it was nearly fully dilated. No drops had been used.

"His vision was 18/20, the dilated pupil faintly responded to light. There was no ciliary paralysis; the iris was yellowish in color, that of the other eye being blue. Both were the same color before the trouble set in. The vitreous showed a slowly floating web which consisted of innumerable dust like opacities and was thick enough to faintly obscure the papilla. When the patient looked straight forward there were to be seen five small highly refractive bodies resembling cholesterin crystals. These were slightly to the center of the vitreous. Nervehead and vessels, normal. Almost directly downward, in the extreme peripheral region of the retina was an area in which the retinal structures had been destroyed. The vitreous in this region showed large numbers of cholesterin like bodies and several large highly refracting strings. There were several small splashes of pigment in the retina adjacent to the area above mentioned. Intraocular tension, 18.

"Nearly two weeks later, Sept. 8th, when I next saw Mr. C., I found the pupil dilated ad maximum; vision, perception of light, the pupillary dilatation and the rapid occurrence in the vitreous of the highly refracting bodies was the combination I had never met with before. I have no objection to your using this letter. You must remember that I had got no history of injury."

At the Library of the Army Medical Museum I found that few authors made any reference to dilation of the pupil in siderosis bulbi. (See A. J. O. v. 6, p. 990 and v. 7, p. 871. Ed.)

Vossius states that he had spontaneous mydriasis in four cases out of fourteen and his examination of literature of this subject has revealed a few similar cases. He attributes the dilation to irritation of the fibers of the sympathetic nerves which supply the dilators of the iris. Following this dilation after a year he has observed a miosis which would not yield to the application of atropin. Atrophy of the musculature of the iris is regarded as the cause of the condition.

With Mr. C. there was a marked dilation of the pupil, irregular on the temple side and slight adhesion to capsule of lens. The adhesion yielded to 3% atropin in one day. Dr. John Dunn states in his letter that the intraocular tension was 18, and he found pupil dilated, and no drops had been used.

The reason I used strong solution of atropin was that when the patient came to my office the tension of the eyeball was about 14 and there was adhesion of the iris to the capsule.

1026 16th St. N. W.

## UNILATERAL NYSTAGMUS.

LAWRENCE POST, M. D.

ST. LOUIS, MO.

The case here described appears closely related to cases of nodding spasm that occur among infants. It was reported to the St. Louis Ophthalmic Society, January 23, 1925.

Rosie May K., colored, aged 8 months, was brought by her mother to the out patient department of the St. Louis Children's Hospital in December, 1924. The baby had had a very severe head cold for two weeks. For the past five days the mother had noted a nodding of the head and a twitching of the right eye.

Family history was unimportant. Past history was that of a normal child. Birth had been spontaneous and not remarkable in any way.

Examination revealed a healthy child except for an occasional rapid backward and forward nodding of the head and a constant horizontal nystagmus of the right eye only, and a coryza with bilateral otitis media. Paracentesis was done on each ear and the child admitted to the hospital.

There was no strabismus; the media were clear and eyegrounds normal. The nystagmus was rapid and of almost equally long components, but at some

times was more marked than at others. The left eye showed no nystagmus.

During the child's stay in the hospital, the nystagmus markedly diminished as the cold and otitis disappeared.

In 1906 Duane[1] collected 52 published cases of unilateral nystagmus. Of these 34 were vertical; 11 were horizontal; 5 were rotary and 2 were mixed. He points out certain features of this type of nystagmus.

(1) Apparent oscillation of objects looked at (true also, sometimes in miners' labyrinthine nystagmus.)

(2) Oscillatory diplopia.

(3) The patient can see the movements of the eye in a mirror.

Because of the age of my patient, obviously none of these characteristics could be determined.

Heimann[2] divides unilateral cases as follows:

(1) Those occurring in very amblyopic and misdirected eyes.

(2) Those occurring in the course of serious nervous diseases.

(3) Others of a transient nature accompanying or following spasmus nutans.

Duane divides unilateral nystagmus into two groups, (A) In infancy and (B) In later life. In infancy it occurs with spasmus nutans and with unilateral opacities in the media. In later life it occurs in unilateral amblyopia and squint, unilateral astigmatism of high degree and in diseases of the nervous system. The cause he attributes to perverted action of the centers governing coordination.

Verhoeff[3] explains cases with squint as follows: "At first there is a cortical lesion, probably congenital in origin, that tends to produce nystagmus of the eyes. This is associated with, tho independent of, an absence of the function of binocular single vision, which leads to the development of squint. Owing to one eye being now used for fixation, its tendency towards nystagmus is fully compensated for. On the other hand, the squinting eye never being used for fixation, its tendency towards nystagmus is allowed to become manifest."

Still[4] has noted that nystagmus associated with head nodding is almost always more marked in one eye than in the other. He thinks that unilateral nystagmus (except in this disorder) is extremely rare in children. It is characterized by its onset without apparent cause in an infant during the teething period, preceded by or following a period of head nodding and further, by its complete disappearance after a few weeks or months.

He observes also that there is usually a very slight oscillation of the eye which is apparently steady. This can be seen if the fundus is viewed ophthalmoscopically.

This was confirmed in our case in which an exceedingly fine vibration was noted on ophthalmoscopic examination, so fine in fact, that its characteristics could not be determined.

The case reported obviously belongs to a group of infants with spasmus nutans. The etiology of the nystagmus is as obscure as that of the concomitant disease. There have been three principal theories of the latter; that it is due to darkness, to rickets and to dentition, but to each of these there are strong objections. The only factor which seems fairly constant is that the winter months, in the Northern Hemisphere, see the onset of by far the majority of the cases.

If we do not know the etiology, we do know that the prognosis is good in this type of nystagmus and hence are able to prognosticate a speedy recovery.

Metropolitan Bldg.

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# NOTES, CASES, INSTRUMENTS

## THE CRAMPTON ACCOMMODATION ROD.

FRANCIS HEED ADLER, M. D.
PHILADELPHIA, PA.

The Crampton accommodation rod is so serviceable that it occurred to the author to combine the rod and the reading cards in one instrument. The apparatus described below was therefore made by Messrs. Wall & Ochs and is so useful as to warrant description.

A metal disc is fitted to slide along the Crampton rod. This disc has an aperture as illustrated, 2½ cm. in diameter, in which can be rotated various sized letters of the reading cards. These letters are mounted on a second metal disc which fits into the first and which can easily be rotated therein.

By this means, as illustrated, the letter card and rod are always together, and the handling of the instrument is much easier than when the letters are separate from the rod.

No originality is claimed for this device, as many somewhat similar instruments have been devised. It is, however, far simpler than most of them and therefore has been found of practical value.

313 S. 17th Street.

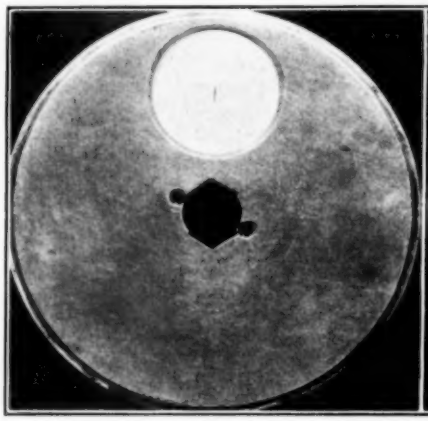

Fig. 1. Showing the metal disc with Duane line in the aperture.

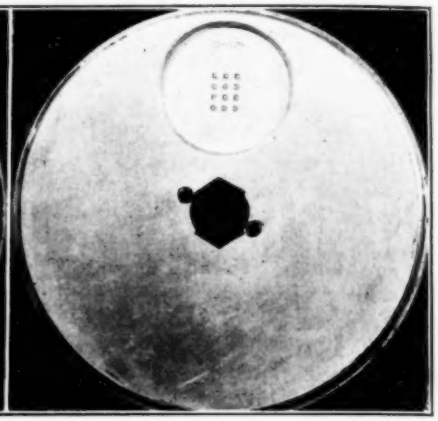

Fig. 2. Showing the metal disc with letters in the aperture.

Fig. 3.—Showing the Crampton Accommodation Rod with the disc in position for use.

## INJURIES TO THE CRYSTALLINE LENS.

W. A. Huber, M. D.

TULSA, OKLAHOMA.

Case 1. Mr. T., aged 33, white, married, consulted me August 25, 1924, giving the following history. Ten weeks ago while working he felt something pierce the right eye. He saw a physician who assured him nothing was in the eye. The sight has been gradually failing, altho he has had no pain, inflammation or discomfort in the eye since the injury.

Vision R., 20/200; L., 20/20. With the pupil of the right dilated I detected a foreign body in the lower part of the lens. This was seen by oblique illumination. There was considerable opacity in the lens also.

August 28. Vision R., 5/200 and the lens was rapidly becoming opaque. The extreme periphery of the lens was still red with the ophthalmoscope at a distance.

August 30. A combined cataract extraction was done. The foreign body was removed without any change whatsoever in its position in the lens.

September 27. Vision with plus 9.50 sph. equals 20/20. Reads Jaeger 1 with plus 3 added. He has binocular vision also, altho the left eye is only slightly hyperopic.

There are three interesting points in this case: 1. Toleration of a foreign body in the eye without any inflammation. 2. Good results of combined extraction of the lens with the foreign body. 3. Binocular vision with aphakic correction. The foreign body was magnetic. This was determined after its removal.

He has been wearing his correction all the time. He has never noticed any diplopia under any circumstances, with normal vision in each eye with correction.

(A few persons with good vision in either eye alone and no strabismus, have alternating but not binocular vision; and cannot perceive binocular diplopia.—Ed.)

Case 2. Mr. W., aged 50, white, married, carpenter, consulted me November 11, 1922, with the following history: Six months ago a splinter of wood pierced the right eye and a workman pulled it out. He could not see for some time. There is a scar in the temporal half of the cornea near the limbus, with temporal border of iris attached (adherent leucoma). The pupillary area is clear, but some opaque lens substance is still present.

Vision, O.D., with plus 11. sph. ⊂ plus 0.50 cyl. axis 135°=20/20. Reads Jaeger 1 with plus 3 added. This gentleman had no operative procedure and received $1,800.00 compensation for loss of the eye soon after the time of injury. This is a case of spontaneous absorption of a traumatic cataract.

New Daniel Bldg.

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## PRIMARY UNILATERAL DIPHTHERITIC CONJUNCTIVITIS.

H. D. Fallows, M. D.

MASON CITY, IOWA.

Believing that this contagion of the eye is uncommon enough to report, I submit the following case, which I have had, in a child 2 years of age. The mother called me because of a swelling of the baby's eye and fretfulness. Examination shows the left upper lid swollen and edematous, overriding the lower lid, with a membrane protruding.

On lifting the upper lid and pulling the lower down, an excoriation of the skin of the lower lid was seen on the margin, extending both ways from the center with a thin grayish membrane on this excoriation. The conjunctiva of the lower lid was very highly injected but no membrane present. Quite a thick grayish membrane was attached to the conjunctiva of the upper lid as far back as the tarsal fold. The cornea was clear. Pupils were equal. The child's temperature was 99.6 with a pulse of 115. Throat and nose were negative. There was no history of sore throat in this child or any of the rest of the family. The membrane was quite adherent to the conjunctiva of the upper lid but did not bleed when removed. Smear and culture were taken at this time.

Examination of the smear showed Klebs-Loeffler bacilli. Fifteen thousand units antitoxin were given at that time. The following morning, the culture was reported positive. Ten thousand units of antitoxin were again given, resulting in the membrane entirely disappearing in four days.

The result is shown in the picture. The cornea and the conjunctiva were clear, but with a depression on the integument of the lower lid on the temporal part. The reason that the integument of the lower lid was involved and not the conjunctiva, I believe, was due to the contact of the upper lid. Mercuric cyanid 1-4,000 sol. was used to cleanse the parts and antitoxin was the only additional medicinal treatment. A Schick test on the remaining members of the family was positive.

Quite a number of diphtheritic con-

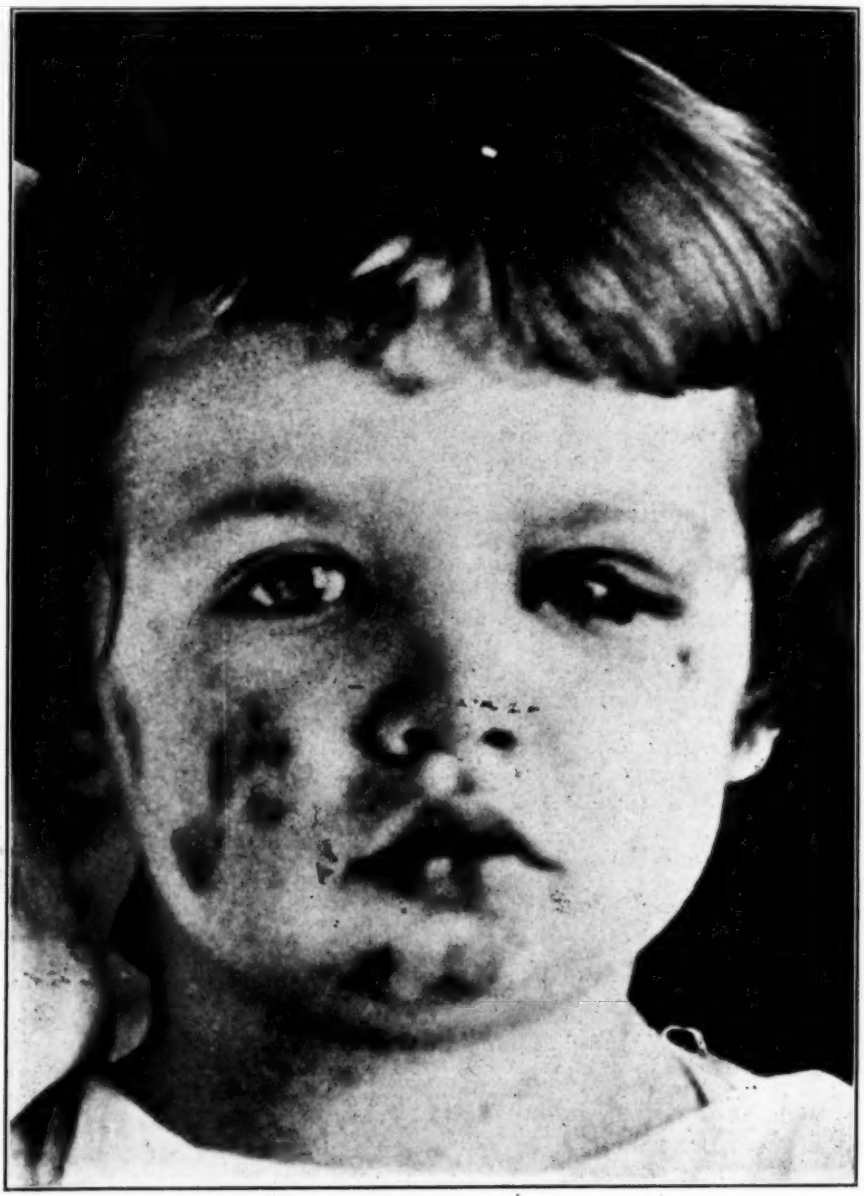

Fig. 1.—Diphtheritic conjunctivitis of left eye with sloughing of lower lid.

junctivitis cases are reported in the French and British Journals but the American Journals have reported very few.

Dr. W. H. Luedde of St. Louis reported a case somewhat similar in 1906. Cases where the infection is primary in the conjunctiva or lid appear to be very rare.

## A CASE OF NEUROEPITHELIOMA.

J. J. HORTON, B. S. B. A., M. D.
EAGLE PASS, TEXAS.

In the United States the majority of the people have reached a stage of development, such that the advice of the physician or quack is sought early for the relief of pain or altered function. As a consequence of these early consultations the patient has a much better chance of being cured. And the members of the profession in the more "civilized" portions of the world have the opportunity to study disease in its earliest beginning, and therefore to learn its cause and to practice preventive medicine. But they miss the pleasure of seeing the end results of disease that has been allowed to develop naturally, so to speak, uninterrupted by science, or at most treated only with the simple and often strange remedies of medicine men or old women. As an example of what a neuroepithelioma can do when not interfered with, I submit the following case history and photograph:

C. C., Mexican, boy, age 5, first seen October 9, 1924. Father has senile cataracts, both eyes. Family history otherwise negative. Mother says that two years ago a "blanquita" or white spot made its appearance on the front part of the eye, and was accompanied with great pain. A few weeks later the tumor began to grow and the pain became less. It had slowly increased in size until now, which was the first time a physician had been consulted. The white spot seen by the mother was probably the cataractous lens pushed forward or the tumor itself presenting in the anterior chamber and encroaching upon the cornea. After rupture of the globe the pain was alleviated.

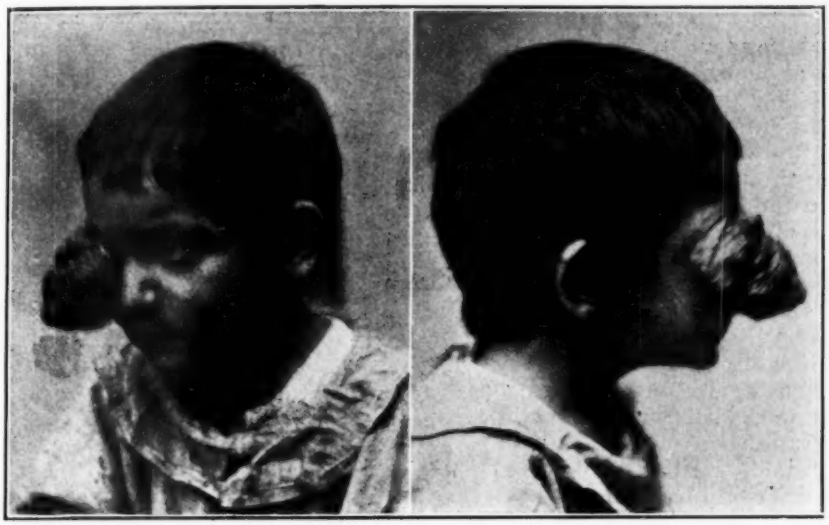

Fig. 1.—Neuroepithelioma of eyeball. Mexican boy aged 5 years showing nasal and temporal views of tumor. 7 cm. from external canthus to apex.

Examination showed an emaciated boy with O. D., a cone shaped tumor occupying the widely distended palpebral fissure and projecting forward. It was covered with old, clotted blood, pieces of green leaves, and live calliphorae vomitoriae. When these were cleaned off it was seen that the lids were edematous and

rather red. They were stretched forward to what appeared their maximum limit. Great force from within the orbit must have been necessary to so stretch them. The margins of the lids, both above and below, were free, except for the outer third of the lower, where it merged indistinguishably into the tissue of the tumor. Most of the cilia were gone. The tumor had become attached to the palpebral conjunctiva about 2 mm. back of the lid margins. Then by pressure from behind, this conjunctiva had become everted, assuming a position anterior to the lid margin. After being cleansed the tumor presented many small bleeding points, and pus exuded in some places. A few fine hairs grew from the apex, leading me to consider dermoid as well as neuroepithelioma as a diagnosis. The growth measured 7 cm. from the forward stretched external canthus to the apex, and the base occupying the palpebral fissure measured 6 cm. in the horizontal by 5 cm. in the vertical directions. O. S., normal.

A complete exenteration of the orbit was done on the same date. The wound was dressed every other day for three weeks, at the end of which time it was looking all right. I did not see the patient again, and I heard that he died five weeks later or two months after the operation.

Dr. Violet Keiller, Medical Dept., Univ. of Texas, Galveston, reported a microscopic diagnosis of neuroepithelioma.

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## THE FIRST BOOK PRINTED ON DISEASES OF THE EYE.

BURTON CHANCE, M. D.

PHILADELPHIA.

Read at meeting of the Section on Ophthalmology of the College of Physicians, Thursday, February 19, 1925.

At a recent dinner of the College, Dr. Keen, in referring to the objects treasured by us, cited "the First Book on Diseases of the Eye," as one of our most precious possesions. It has occurred to me that it might be of interest for us all to know something about one of the milestones of our Art, which was laid eighteen years before this Continent was discovered, a copy of which now, four hundred and fifty years after it was published, reposes in our Library.

The book is entitled—"Benevenuti Grassi Hierosolomitani Doctoris Celiberrimi ac Expertis Simi de Oculis Eorumque Egritudinibus et Curis Feliciter Incipit." It was printed in Ferrara in 1474, and, therefore is ranked among the first books printed by movable type. It is probable that there are but two other copies in America, one at the Surgeon General's Library, the other in the Morgan Library. Because of its very great rarity it possesses a sentimental as well as a medical historic value.

The author is listed in the Surgeon General's Library Index as "Grapheus, Benevenutus." His name, however, can be found in many forms—Grassus; Bienvenu Raffe; Benevenutis de Jerusalem (as expressed in the title of our book); Binimitus Grafton; Benevenutus; and Ben Vengut de Salern. Under one or other of these names he was known as the most famous oculist of the Latin, European, and Christian, Middle Ages. The time and place of his birth and his death are unknown; Hirschberg is inclined to place him in the XII century, but there is greater certainty that he did not flourish before the XIV; Guy de Chauliac, in 1363, knew of him. He is believed to have been a Jew, of Jerusalem, while it is known that he studied at Salerno, and that he practiced in Italy and the south of France.

His manuscript treatises "Practica Oculorum," and, "De Morbis Oculorum" were for centuries standard works thruout Christian Europe, and copies of these treatises in the early western European languages, as well as in Latin, are still extant. This printed book of 1474 is a revision of the earlier treatises. It contains the ophthalmologic science of the ancients and also of the Arabians. It possesses little of original value, but is rather an epitome of the accepted methods of the practitioners of the author's era; based upon the writings of the ancients, which

were well known to the physicians of western Europe, as well as upon those of the Arabian writers.

The book consists of 70 pages of twenty-five lines each—the type being rather heavy, yet beautifully clear and perfectly aligned. There is no title page, as is usual in the early books, and the printer's name is abbreviated below the last line of the last page. Below the name are two "F's" and four "I's" which are believed to indicate that it was "completely finished" in the year "1474," the printer having used similar signs in other books whose dates have been certified as of years earlier and later—as for instance "F——F——II" and "F——F——V" being respectively, "1472" and "1475."

The intimate history of our volume is not known. The binding is not the original covering. On the margin of several of the pages are notes and corrections written in an antique hand in Latin. At another time it may be that I will tell more in detail concerning the book.

So precious is our copy, for it is one of the first edition, that it is not allowed to one to take it away from the case where it is kept. It may be seen at any time by anyone on request to Mr. Fisher. A photostatic copy has been made, however, and this may be freely consulted.

1305 Spruce St.

# SOCIETY PROCEEDINGS

## PITTSBURGH OPHTHALMOLOGICAL AND NEUROLOGICAL SOCIETIES.

JANUARY 26, 1925.

DR. T. M. T. MCKENNAN, presiding.

### Fundus Changes in Conditions of Intracranial Pressure.

DR. EDWARD B. HECKEL. The late Dr. Noyes said to me, that external diseases of the eye are usually learned within a very short time; whereas diseases of the fundus are a lifelong study. I have never forgotten that. It has impressed itself on me more and more. No matter how many thousands of eyes may have been observed, the newest one always offers new problems which tax the skill of the observer.

It is wise to remember that the viewing of the interior of the eye is quite an art, as well as a science, and one of the first things to remember is, we sometimes miss certain things about an eye by attempting to make an ophthalmoscopic examination of the fundus, without any preliminary survey of the organ as a whole. It is wise to take a bird's-eye view of the eye before getting up close to look into it. For instance, I recall the case of a distinguished gentleman in this town who had made the rounds of a number of men, and made many trips to laboratories for urine examinations, etc. The man was supposed to be suffering from a retinitis. He called on me and I noticed his pupils were widely dilated, he had just gone thru with another ophthalmoscopic examination. In the dark room, at a distance I examined the lens of either eye. Both lenses were decidedly misty and cloudy, and on closer view of the fundus I found that it was slightly hazed by this condition of the lenses. I made up my mind after viewing each eye carefully that his fundi were negative. He started to tell me something. I told him not to say anything, that I would tell him what I thought and he could draw his own conclusion. I gave him my opinion that the fundi were negative and that the lenses were hazy. Some weeks later I learned what happened. He went to New York, consulted one of our eminent oculist confreres there, who told him what I had told him, and returned home very much satisfied.

Viewing the fundus is very much like viewing a picture. One man will see much in a picture that another man will not see. Often it is a matter of opinion, and sometimes of powers of description to tell another person just exactly what one does see. Tonight

the discussion limits itself practically to conditions of the fundus that may be indicative of intracranial pressure. I think the only condition that is supposed to indicate that is a typical socalled papilledema.

In a papilledema, the rest of the fundus remains perfectly negative, is clear and often may be normal. To some men it seems rather startling, there should be an elevation, sometimes of even five or six diopters, and the rest of the fundus be clear. In retinitis, there is an inflammatory process; and in all inflammatory conditions of the retina and head of the nerve the first thing that happens is that the retina loses its transparency, so that ordinarily we do not have much trouble differentiating between retinitis and a retina that is practically normal. If you have a retinitis of any type at all, you will have more or less of a haziness, or an edematous condition. We must not lose sight of the fact that there are a number of conditions which are practically normal, in fundi which vary from the typical fundus.

An elevation of the optic disc in a typical papilledema is over two diopters if it has gone on any length of time. If it is just starting, of course, it may be very slight, but the rest of the fundus will be perfectly clear and distinct.

The appearance of a pure, true papilledema, in itself is not indicative of an intracranial neoplasm, or particularly of intracranial pressure. We may have a nephritis, that is complicated with typical papilledema, which may run on for an indefinite length of time, without any changes in the retina itself, and cases have been reported where it has gone on and the retina has been observed to show no change to within two days of a fatal issue.

We had a case last fall in the Allegheny General Hospital that came in under the neurologic department. A young girl had had excruciating headaches at times, projectile vomiting, and practically all the other signs of a so-called tumor. Her fundi showed a papilledema of about five diopters, if I remember rightly, and the rest of the fundus was negative. She went on for a while and the neurologic department said it was not a tumor. Now, she has nephritis with other signs and symptoms characteristic of this disease.

You must also bear in mind we have a socalled pseudopapilledema which exists occasionally. I have run across one case in this city, and I have tried to get hold of the patient on several occasions. The picture of pseudopapilledema differs from the picture of true papilledema in that in papilledema there is obstruction of some of the vessels. In a pseudopapilledema, the vessels come up and roll over and then pass off into the rest of the fundus, and they are perfectly clear and distinct. It looks very much like the water of a fountain that rises up thru a basin, and is turned down just low enough to have the water rise for a little distance and fade off in the basin. Those cases do occur. But the chief thing to remember, and what I would like to emphasize for discussion, is the fact that a papilledema in itself is not pathognomonic of intracranial neoplasm. You may have it, as my neurologic friends so well know, with an edema of the coverings of the brain, the meninges.

Dr. John B. McMurray. First of all, we must differentiate, if possible, between the socalled papilledema or passive noninflammatory swelling of the nervehead as it occurs in intracranial pressure, and active inflammation of the nerve as a result of retrobulbar inflammation, or inflammatory changes of the nervehead within the globe itself. If our conception is correct, that the changes at the nervehead are due to intracranial pressure, there must be a certain sequence of events, that would help us to determine whether, or not, the condition observed in the fundus is the beginning of socalled choked disc, due to intracranial pressure, or whether it is due to one of the true inflammatory changes.

The first change, possibly, is edema. Dr. Cushing has emphasized very much the early picture of choking, and he particularly emphasized the peripapillary edema as the very beginning stage of intracranial pressure. I think the

second important change is venous stasis. The change in the veins is one of the most marked, in the early stages, observed within the eyeball as a result of the intracranial pressure. The other day a case was brought to my attention of brain abscess, temporosphenoidal lobe, from a chronic ear lesion. The patient was seen in the morning. During the night she had had some convulsions, was in a semicomatose condition at time of examination. The fundus, left eye, showed veins very full, arteries not changed, disc edge somewhat blurred. In other words, I felt as though in three or four hours, or during the day at least, a full fledged choked disc would develop. At noontime the same day she regained consciousness and much to my surprise, on reexamination of the eye, the venous stasis and edematous appearance had all disappeared.

Whether or not that means anything, I believe we ought to examine the fundus of suspected cases of intracranial pressure more frequently than we do, that we may observe the changes occurring in the venous circulation, indicating interference with venous return as a result of pressure in meningitis, in abscess and also in beginning of brain tumor. So, I would emphasize these two points, that where we are dealing with a noninflammatory type, such as choked disc, there must be, if our conception of its cause is correct, edema of the retina adjacent to the nerve, with obscuration of the disc edge and changes in the size of the veins. The arteries are not affected at the beginning but the veins are full. When this condition exists, it is the beginning, or early sign of choked disc.

After you have marked swelling of the disc itself, with hemorrhage occurring along the course of the veins, with occasional wooly patches such as you find in nephritis, the picture is much complicated and it is very hard, as Dr. Heckel has said, to differentiate between inflammatory or toxic changes, and those changes occurring late in choked disc. I believe, however, if we will study our vessels a little more carefully, particularly in the early stages, we will be able, with the ophthalmoscope, to help differentiate between those changes due to nephritis, toxemia, etc., and those changes occurring as a result of intracranial pressure.

We all know there are two theories regarding the cause of choked disc; one calling it descending inflammation and the other pressure itself. I cannot understand how a descending inflammation can produce the changes occurring in increased intracranial pressure. The appearance can be explained by strangulation of the lymph and venous return. Just why it is that we get a larger percentage of choked discs in children or young subjects, than we do in older patients, I cannot explain satisfactorily to myself, but almost all neurologists make that statement. They say further, in tumors of the brain what we call choked discs are found in only about 84 per cent of the cases.

In those instances where choking is observed earlier in one eye than in the other, Dr. Parker has suggested that it will be earlier in the eye with the least tension. It is possible for an eye to have a few millimeters difference in pressure and yet not be in any sense glaucomatous. He says himself that this observation needs more proof to establish it as a fact. Also eyes show choking a little earlier if hyperopic than if myopic. This is a fanciful theory based on the angle that the sclera forms with the entering nerve. A myopic eye enters at a more obtuse angle and a greater amount of pressure is necessary to produce stasis of the lymph and venous return. Dr. Heckel has mentioned about the mistakes that can very easily be made in interpreting changes in the study of the nervehead, and does not believe we should rely on the appearance of the picture alone. We should have all the facts before us—refraction, neurologist's report, laboratory tests, etc.

DR. E. E. MAYER. The neurologist is often required to look at the fundi and often he is not able to interpret correctly what his eyes see. It is to his confrere, the ophthalmologist, that

he should and does turn for enlightenment as to what the ophthalmoscope reveals. Yet he has a distinct and separate function in utilizing the fundus picture, in correlation with the presence or absence of other symptoms of brain involvement. He is concerned with intracranial pressure in general, rather than with papilledema in particular.

Since with choked disc, we are dealing with an effect of intracranial pressure, one must reason out how the pressure produces a papilledema and also why there is a divergence of opinion as to whether other diseases than brain disease bring on this symptom. It will occur occasionally with severe anemia or with nephritis. We cannot disregard the inference that in some unexplained way brain edema or venous stasis comes on in these diseases and a papilledema results. I can not believe that there is any other way to cause choked disc except thru increased pressure.

The pressure effects vary, of course, according to the part of the brain which is primarily affected. The pressure effects do not simply result from the fact that the brain is in a closed cavity and has only a definite space in which to spread. The degree of dilatation of the ventricles and rate of increase and absorption of cerebrospinal fluid is a more important factor. Whenever the foramen of Monroe, or the aqueduct of Sylvius, or the foramen of Luschka becomes closed, a rapidly increasing choked disc is to be expected, unless the patient is young and can acquire an external as well as an internal hydrocephalus. In cases without cerebrospinal fluid accumulation in brain growths in children, the springing of the sutures often saves them from papilledema.

The variations in incidence of choked disc depending upon the nature of a brain growth and its seat are well known. Midbrain tumors may go along until death without any papilledema. Cerebellar growths almost always show a papilledema. Gliomatous neoplasms do not tend to bring on a choked disc as early as does a fibroma. The importance of ventriculographs in suspected brain growths has been emphasized as you know by Dr. Dandy. The work of Batten and Collier also, in interpreting symptoms of compression of the posterior roots of the cord as due to increased cerebrospinal fluid pressure when found in suspected brain tumor cases, is a noteworthy advance; and our diagnostic failures in interpreting localizing symptoms do not ruffle our equanimity as much as before their report appeared.

On the surgical side, the quieting down of a papilledema after opening the skull cavity, and the frequent hernias developing, certainly attest both to pressure being a primary factor, and to accumulation of fluid in the ventricles from closure of the gateway being a prominent secondary factor. Oldtime explanations, failure to note pulsation of the dura, or loss of capillary arterial circulation as a cause of papilledema, I do not believe are acceptable today.

I defer to the opthalmologist in differentiating papilledema from optic neuritis. Saenger and Wilbrand and other writers claimed that they represent two phases of the same condition, in that a neuroretinitis is always present when you find a papilledema; only covered over, as it were, by the choked disc. Or is it true that a long continued choking, only, is followed by (not accompanied by) a retinitis and a consequent atrophy later?

We are often unable to find evidence of focal signs in brain tumor, and neurologists often fail because of this fact to interpret during life what is going on in the brain. I recall for instance a patient who had several transient epileptiform seizures, with the subjective complaint of occasional diplopia. I found no evidence of brain disturbance. Dr. Markel went over his eyes and also reported negatively. Yet the young man died two weeks later from a midbrain tumor.

A neurologist's error may, in connection with the fundi, be of the opposite nature; seeing, because of high refractive error, a papilledema where none exists. I recall a case of this kind in

which I was checked up by Dr. Krebs, who pointed out this fact as the reason for my poor ophthalmoscopy.

DR. GEORGE J. WRIGHT. It is my habit to make a study of the fundi. I have learned, in the course of time, to do this from the neurologist's point of view for three reasons. In the first place, in the routine examination of certain kinds of cases, it is of enormous value to know at the very beginning whether or not there is any abnormality of the fundus. I think it does not take very long for a man to recognize that the fundus is normal or near normal, or at least not suspicious. In certain cases that is a good place from which to start.

The second reason is I feel that we ought to study the fundi constantly, as we do the knee jerks and the like, because the fundus changes are variable and occur at different times. A case of tumor suspect may, in the beginning, show absolutely no fundus changes; and, in later study of the case, the suspicion of an increasing intracranial pressure may be eventually confirmed. We cannot in hospital practice, or in office practice, have the ophthalmologist always at hand. When fundus changes begin and are, in my judgment, definite, then I like to have the ophthalmologist come in, and say whether or not my own observations are correct.

The third reason why I think the neurologist should be in constant touch with the fundi, is that if there is anything there, he may from the neurologist's point of view, have facts or information regarding the situation which at least he can discuss, humbly perhaps, but at least discuss, with the ophthalmologist. I think there comes a time, as has been indicated tonight, when the question is a debatable one; it is not always well for the neurologist to have a fixed opinion without being checked up. And it is not well for the ophthalmologist in his examination to write down a certain thing and let it go at that, without having a discussion of the situation in the light of the other problems, or of the other symptoms, that may be present in that case; and therefore I feel that a careful study of the fundi by the neurologist is almost a necessity for careful and satisfactory work, especially so in a condition that is steadily progressing to an issue.

Some time ago one of the men working at Johns Hopkins brought up this question of the nomenclature in fundus changes. I tried today to find the article but I could not put my hands on it at once. It refers to this question of the difference between neuroretinitis and papilledema which has been mentioned here tonight, the fact that sometimes it is difficult to decide which is which.

The neurologist, I think differentiates neuroretinitis from papilledema. It is a distressing thing for the neurologist when he sends a case for an eye examination to have the report come "neuroretinitis," when he expects the ophthalmologist to say "papilledema." If they get together sometimes it is found that really the ophthalmologist is leaning toward a papilledema, or feels it may be a papilledema but doesn't care to say so. "Papillitis" has been used as the general term and neuroretinitis and papilledema as two forms of papillitis. It seems to me that if a case is debatable it is better for us to have the term papillitis used, and wait for further symtoms before declaring that it is a neuroretinitis, or an optic neuritis, or a papilledema. I would like to have some expression of opinion as to whether or not that nomenclature is right or proper.

The question of choked disc is one that of course means to the neurologist, chiefly or entirely, an increase of intracranial pressure. The most common cause of this is tumor, and in the ordinary case that is what we think of. Choked disc also occurs in certain cases of meningitis, because of the block that is produced in the narrow aqueduct of Sylvius or in the foramina of Magendie and Luschka. It seldom occurs, yet it does occur, and I always look at the fundi as a matter of routine. Choked disc also occurs in epidemic encephalitis and in nephritis.

I remember a case in which I was the neurologic consultant with a well known internist; and in the routine ex-

amination of the eyeground choked disc came up, and I said that the nephritis which was present was entirely sufficient to explain it. He was very dubious about it. Being a much older man than I, I was anxious to impress upon him that this thing had been found and reported by others. I found an article by Dr. Collins and another by Dr. Spiller, and read extracts from these articles to him over the phone. I made my point, which was afterwards substantiated by an autopsy, in which no tumor was found.

For reasons that we do not quite understand, choked disc occurs in encephalitis or sleeping sickness, not commonly but definitely enough to make the thing of great importance. The differential diagnosis between encephalitis and tumor is a very important one, not only as regards prognosis but as regards treatment. The occurrence of choked disc in sleeping sickness, has been repeatedly reported. Dr. Spiller has had three or four cases. The position of the neurologist in a situation of this kind is an extremely anxious one, especially if the ordinary signs of encephalitis are not very definite. I understand the degree of swelling in encephalitis is not high, rarely above two and a half to three diopters, and perhaps that may be of help in diagnosis.

Trauma also causes choked discs, and by this I mean trauma that is not necessarily associated with massive hemorrhage. Trauma with massive hemorrhage of course has the same mechanism as tumor. There have been some cases reported, (I have three or four of them myself), in which, following what seems to have been an ordinary concussion, the late development of a choked disc is a possibility and an extremely serious one. Dr. Weisser can testify to this, as we both have record of a young girl who was operated on at the Mercy Hospital for removal of the tonsils, fell out of bed in the confused state after the anesthetic and bumped her head. In a few days she went home but complained of headaches for a month, and nothing was done about it. It was looked upon as a simple thing at first. After she began to lose her sight, she was taken to the doctor who had operated on her, and he shifted her over to Dr. Weisser who found an enormous choked disc with almost total blindness. She was then referred to me and I didn't know what was the matter at that time. It seemed to me inconceivable that such a slight accident would be a sufficient cause, but within an hour a decompression was done.

Here was an enormous choked disc, vision was practically gone, and I had only one trick left, and that was to advise a decompression. Her spinal fluid was examined after operation and we found nothing. The choked disc went down, and that child is alive today with practically normal vision. I have had a similar experience three times since; twice in boys that were in football games, and had the ordinary bump on the head with signs of a mild concussion. In one instance the situation was very, very trying for the doctors. We had before us this problem: Had he a tumor, or was this accident which we knew about and which was immediately followed by a train of pressure symptoms sufficient to explain his condition? In this particular instance we resorted to very careful manometric studies of the spinal fluid, which showed increased pressure. We drew off a little fluid several times in the course of two weeks and that settled it. The boy is well today. I mention this because of its importance and because of its possible serious results.

Dr. Heckel indicated that choked disc might also occur in serous meningitis. In the St. Francis Hospital we have cases of the kind occasionally, in alcoholics. If I saw a choked disc under such circumstances I would not be surprised; it would not worry me; I would think the problem was the problem of getting an alcoholic well. But in a considerable number of cases, aside from mere overfullness of the veins, I have never seen a choked disc.

Another thing I want to speak about, is the possibility of choked disc being associated with a sinus infection. I cannot myself answer that question. Somebody in the literature some time ago started that problem and it

bothered Dr. Cushing so much that I believe he felt constrained to write an article, and warn doctors, rhinologists and neurologists against a too easy interpretation of a choked disc on that basis. I should think, from my own experience, that sinus infection would be a good cause for a neuroretinitis; but that it could cause what would be a typical choked disc I have doubt. In any case of suspected brain tumor with a choked disc, in which the other signs of tumor are very definite, I do have a careful study made of the sinuses and have been prepared to interpret what is found cautiously. That the sinus infection is the cause of what seems to me a choked disc I would hesitate to make a final judgment.

I was interested to hear Dr. Heckel speak about pseudopapilledema. A case I had had caused me enormous worry, because I thought the patient had a choked disc, due to tumor, for she had a terrible headache. The eye man reported that it was a pseudopapilledema. He said it sometimes occurred with high degree of hyperopia, and that the headache also could be caused by that. It had to me the appearance of a true papilledema, which it was not.

In the question of neuroretinitis we have to deal with the local or general toxic cause. I think Dr. Stieren will remember an incident in which a patient had what seemed to be a choked disc and we tried to make a brain tumor out of it. Shortly afterwards this woman developed a local neuritis in the arms, which seemed to us a signal that there was an underlying general infection. I have understood that the patient has since got well. What looked like choked disc was probably not a choked disc at all.

One of the most distressing things is to see at autopsy a large tumor of the brain with so little neurologic evidence of its presence. I don't know whether ophthalmologists appreciate how difficult it is sometimes to get much information to prove that there is a tumor. We don't need much. In a case of papilledema, if the reflexes are distinctly unequal, or if there is a Babinski, that is a great deal; and yet I had in the Mercy Hospital, at the same time, two cases of undoubted tumor without any objective signs except choked disc. They were operated on for decompression and enormous pressure was found. A tumor may be present and give very little data. If we look over a series of Dr. Cushing's reports, with the data on which he diagnoses the tumor and its localization, a few words or a few lines cover the whole thing and not a mass of data. The examination must be carefully and discriminatingly made, but it is a very difficult and unsatisfactory thing as a rule. In possibly only forty per cent of the cases a localization of tumor can be made. That is why such aids as ventriculography are of great help sometimes to run a problem down to a conclusion.

*Discussion.* Dr. Edward A. Weisser. Relative to the terms papillitis and papilledema, I am of the opinion that papillitis is a very bad term to use. It does not signify anything but an inflammation, be it from the eyeground going up or some inflammation from the brain coming down. I think it is better to signify what we mean, in speaking of an inflammation of the optic nerve, as a neuritis, and then say it is either an ascending type or a descending type. If I were to give an opinion as to the type and stage, I believe it would be better to divide it into stages of inflammation as viewed by the ophthalmoscope; then say whether it was thought to be an ascending or descending neuritis.

Speaking of pressure symptoms of descending neuritis. The first stage shows the disc blurred and the excavation practically filled in, the arteries very small, the veins quite large and tortuous, not much change in retina or loss of vision. In the second stage we find the disc swollen and choked, the arteries thin and some occluded, veins larger, edema of the retina around the disc. It is in this second stage that the disc shows some abnormal height and the term papilledema is used.

In the third stage we observe the degenerative changes, exudates and hemorrhage subsiding and absorbing, the disc itself becoming a dirty gray

color, the physiologic excavation being entirely filled with connective tissue, and later the entire disc may go on to a complete secondary optic atrophy.

I would like also to say something of the case Dr. Wright reported. If I remember correctly she had no injury whatever. That case was treated for spinal meningitis. She was going blind before she came to us.

Dr. Edward Stieren. Mr. Chairman, this joint meeting has meant so much to me that I took the time this afternoon to go over my case records of brain tumor. I have complete records of thirty-six cases. That means about a case and a quarter a year and, as we have in the Pittsburgh area about thirty trained ophthalmologists, if the same proportion holds it means that there occur in this district, with approximately a population of 2,000,000 souls, about thirty cases a year, which gives sufficient material for considerable study of choked disc in increased intracranial pressure.

The picture, of course, is one of edema; it is not one of inflammation at all, and my conception of the mechanism of this edema is a little different from what I think is generally accepted, that there is sufficient pressure of cerebrospinal fluid within the skull cavity to force this fluid thru the arachnoid into the sheath of the optic nerve. I cannot conceive of any pressure high enough to do that, and the patient still live. I think the more probable way it occurs is by direct pressure on the return circulation, in the optic nerve and from the disc. The pressure in the arteries is about one hundred millimeters of mercury, and in the veins and lymphatics it is hardly one-tenth of that, so that the first thing that occurs is the damming up of this circulation. You get a water logged nerve and this is the picture you see at the optic end of it.

Dr. Wright brought up one very interesting point, when he said that Dr. Cushing has given some study (and I know he has) to what might be called sinus papilledema, where there is an involvement of any of the sinuses accompanied by choked disc or inflamed nerve.

One of the characteristics of papilledema is that central vision is rarely diminished at first, but in these cases of optic neuritis which occur with sinus trouble, almost the first symptom is reduction of central vision, and my experience is that it is very rarely recovered, if inflammation goes on, as it usually does, before the condition is thoroly recognized. I think that in these sinus cases we have to deal with a toxic neuritis from the neighboring sinus.

In regard to choked disc and lethargic encephalitis, Dr. Wright said I was quoted as having said I had seen such a condition. I do remember the case now, the patient being a child that had a low grade choked disc and made a complete recovery. In regard to the other case he spoke of, I was suspicious of the true nature of the papilledema for the reason that the woman had a very much constricted field; and she has to this day. The swollen appearance of the nerve has gone down, but she has a restricted field without any pathologic findings in the retina, and I had to regard it at the time, and still do regard it, as a case of hysteria.

Dr. Mayer—in speaking about the incidence of choked disc, in regard to the site of the tumor; I find in my case records that cortical and subcortical neoplasms, tumors, or growths, are less apt to give choked disc, than those in the region of the pons or in the cerebellum. The first case of trephining for the relief of choked disc I witnessed when a student at Johns Hopkins. The moment the meninges were incised to give vent to the fluid, the patient went into sudden and absolute collapse and never recovered. Hence I believe it is well in cases of tumor near the base of the brain, or in the cerebellum, that the fluid be drawn off very gradually so that this sudden depression does not occur.

Some years ago one of our internists, whom we all know well, felt that an ophthalmoscope was very important and used it with great enthusiasm. He called me in the ward one day to see a patient he contemplated having trephined and said, "He has all the

symptoms of brain pressure and an enormously choked disc. Just look at this. Here is a plus seven in the ophthalmoscope." It is true that disc was seen most plainly with a plus seven lens, but he had seven diopters of hyperopia; and when he had glasses put on the vomiting and headaches disappeared. As to the side of the brain of the tumor, being told by the heights of the papilledema on each side, I don't think we can place any dependence on this. Some years ago in a case that finally came to autopsy and proved to be a large glioma, the disc was enormously elevated on the right side and the tumor was found on the left, so there is nothing in that to rely upon. There is one picture in the fundus which invariably accompanies papilledema, and that is displacement of the retina laterally, which can be seen, especially with red free light by focusing on the retina.

Dr. George H. Shuman. I should like to stress an aspect of this important subject which has not been touched upon by any of the previous speakers. I refer to the role that subjective visual signs and symptoms and functional testing of the faculty of vision plays in the diagnosis and prognosis of conditions associated with intracranial pressure. The earliest manifestation of an oncoming papilledema may be transitory attacks of blurred vision, or even blindness lasting from a few minutes to an hour or more. A high degree of papilledema may be present, without either the visual acuity or the visual fields being affected. Nevertheless, I do not think that an opinion upon the ophthalmologic aspects of a case, especially if it be a borderline case or one difficult of diagnosis, should be considered complete or entirely trustworthy without a record of the visual acuity, a careful study of the fields for both form and color and for scotoma, the measurement of the blind spot, and a determination of the light senses by some method of photometric examination. Particular attention should be paid to determining the color perception, especially for blue and red, for defects in the field for these colors may occur in advance of any impairment of central vision or peripheral sense for form (de Schweinitz). In making these functional tests, all the modern refinements of technic should be used, by a person thoroly trained in the work. Very often a knowledge of other aspects of ophthalmology is quite necessary in order properly to interpret disc or fundus findings.

Dr. C. H. Henninger. I am still at a loss to know about the indication for decompression, whether the degree of choking or the rapidity of its development should determine when a decompression should be done, in cases where the tumor is not localizable.

Dr. McMurray described a case where, following a convulsive seizure, the venous congestion was so marked that it gave the impression of beginning choked disc but that it disappeared very rapidly. This has been of interest to me for a number of years, because at one time I had an opportunity of studying a good many epileptics, and found the pupils dilated by the convulsion and the ophthalmoscopic examination easily made. The convulsion produced a marked engorgement of the blood vessels of the retina, and in status epilepticus this sometimes looks very much like a choked disc. This congestion disappears very rapidly with the return of consciousness and the normal activity of the individual. A case that had repeated convulsions had chronically dilated veins of the fundus.

Dr. W. H. Mayer. Dr. Heckel's case was seen by me, and tho to my unskilled eye the picture was one of a typical choked disc, I could not find enough in the history of physical examination to justify a diagnosis of intracranial tumor. I was amazed that this child was practically without any visual disturbance, in spite of the intensely choked disc. Later I understand that the picture was one of typical neuroretinitis.

In regard to springing of the sutures preventing papilledema, I have had a typical Rolandic tumor with an intensely choked disc with actual separation of the cranial sutures in a child of eight years.

CHAIRMAN MCKENNAN. I have a case, operated on last week, of a woman who had possibly a brain tumor. She has, however, a choked disc of the left fundus only, with almost no focal signs. I had my neurologic friends go over her, and we all reached the conclusion that there was something, probably subdural and likely a tumor. Her condition was getting worse, and the eye condition especially was advancing so rapidly that I considered finally that it was probably best to have her decompressed. I took the advice of Dr. Henninger, Dr. Wright and Dr. Mitchell on the subject and they all agreed. She had a decompression on the left side over the occipital lobe. I didn't want to go too far, I was afraid, and I think the results justified that fear. I believe if anything more had been done she would have died. At all events, Dr. Meredith opened in the region of the occipital area, and made a good sized opening, and there was quite a bulging of the cerebellum; but no fluid came out upon opening the dura. Dr. Meredith put a trocar in the lateral ventricle and a beautiful fountain of fluid came out under considerable tension. I might say that she had several spinal punctures, and the fluid was under no tension whatever in the spinal punctures; showing, I think, there was some blocking, probably at the base. At all events, as this fluid came out down went the tension on the cerebellum. Dr. Meredith probed the cerebellum with a fair sized needle to see if he could strike anything that seemed of a different consistency and he could not. I urged him to go no further and he closed it up. We may possibly have a secondary operation if anything further develops. She almost died, the shock was so great. I believe if she had been kept under anesthesia much longer she would have died. But she is very comfortable now. She had had a good deal of vomiting and headache. Her headaches, tho, were confusing to me. Sometimes she would complain of headaches, and then at other times she would have no headaches at all, and that is not usually the case with brain tumor, those generally have pretty consistent headaches.

Now with regard to pressure, especially of the cerebellum fluid, I can recall a case that I had about two years ago, of a man who had very severe epileptic attacks, so severe that I was afraid he would die. At the same time he had the most intense headaches, almost every day; and he had two years prior to that a cranial injury, with removal of some bone that was depressed. The X-ray disclosed a trephine opening. It didn't show anything else, but I felt that there might be some hemorrhagic cysts in that region, the result possibly of the injury; the cysts arising after the closure of the wound and not giving evidence for some little time as sometimes occurs. I have seen it before. So I had this opened, with the hope that we might find some cyst, but there was none. The brain did not pulsate at all when it was opened. Dr. Meredith did this operation on this man. When he opened the membranes there was a great deal of cerebrospinal fluid seeped. It did not come out with a gush like this fountain that came out of the lateral ventricle, but it seeped out for a long time and the brain was quite edematous. That man is still under my observation. He has had no epileptic attacks since. I am curious to know what is the pathology. Some of the bone was removed originally; and there was no pulsation there at the time of this operation and now there is pulsation, which I think ought to be unless the fibrous tissue becomes so dense that it stops the pulsation so that you cannot feel it. I recall that we had the ophthalmologists look at his fundus and they did not find any abnormal condition. I have examined his fundus since and saw nothing. But here was a case that unquestionably had intracranial pressure from increased cerebral fluid, in which there has been no return of any pressure, since, and I am unable to tell the pathology.

DR. EDWARD B. HECKEL. I have not much more to say, except to add that I think the ophthalmoscope should be part of the armamentarium of every

neurologist. He should learn to use the ophthalmoscope and learn to use his own eyes. I think the ophthalmologist should rely on his powers of observation first, and then on the history. I think we should learn to recognize what we see and differentiate it, the normal from the abnormal. I often think that in our modern methods of teaching medicine, too much reliance is placed on what we have on paper instead of cultivating and educating our own senses. We neglect that and we should especially cultivate the power of observation.

Now, the first symptom we recognize is papilledema. There is a venous stasis. Just exactly how that is produced is still sub judice.

It has been our good fortune, in a number of cases, to pick up a papilledema, even when there was no external evidence of any injury, and advise decompression. The decompression was done and the papilledema disappeared. As to the question that Dr. Henninger brought up, as to when to do a decompression in the case of papilledema or socalled "choked disc"; if it is the result of injury or trauma, the sooner the better, because there is only one outcome and that is optic atrophy. The decompression at least should be done, the sooner done the better; but we should not lose sight of the fact that we may have practically a typical papilledema which may not be associated at all with an intracranial neoplasm, and also bear in mind a few of the other abnormalities which would indicate something else.

Dr. John B. McMurray. We have narrowed ourselves down in discussion to the intraocular appearances that occur as a result of increased intracranial pressure so that there has been no occasion to discuss the differential diagnosis of choked disc, or papilledema, and other allied conditions. Of course, that should be taken into consideration and I personally feel that no one should base his diagnosis upon the appearance alone. As Dr. Wright has said, it is always a good thing to take the lead suggested by the appearance of the fundus, and then prove or disprove whether it is a papilledema or a papillitis. These two conditions are so distinct in my mind that we ought not to use the term papilledema when we mean papillitis, or papillitis when we mean papilledema, because they are two distinct conditions. One is a general systemic condition, toxic or otherwise, and the other is a stasis and non-inflammatory.

Dr. E. E. Mayer. I intended to convey the idea that the old viewpoint as given for instance by Bergmann and von Graefe, that papilledema was due to the inability of the brain in its closed box to stand compression, was faulty, because it did not take into account ventricular dilation or closure of the various foramina thru which the cerebrospinal fluid (secreted constantly) passed to reach the spinal canal.

Dr. Wright's discussion of encephalitis reminds me of the diagnostic value of the study of the sugar content of the cerebrospinal fluid; often increased in encephalitis, normal in brain tumor, and decreased in cerebral lues. It has not helped me but is given by some clinicians as being of diagnostic value.

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## BALTIMORE MEDICAL SOCIETY.

### Ophthalmological Section.

March 7th, 1925.

**Corneal Disease.**

Dr. Jesse W. Downey presented a case, giving the following history: For fourteen years there has been repeated corneal ulcers in both eyes, leading in one eye to complete leucoma. There are scars in the conjunctiva, but no typical trachomatous granulations. There was marked shrinkage of the conjunctiva of the lower culdesac. The conjunctiva of the upper lid presented no signs of present or past trachoma. He was unwilling to make a diagnosis.

**Pemphigus.**

Dr. C. A. Clapp presented a case of a middle aged woman with advanced pemphigus of the conjunctiva in both eyes, the cornea of the left eye having been completely covered. There were also definite signs of pemphigus on the soft palate.

**Slit Lamp Examination of Lens.**

MR. BASIL GRAVES, of London, read a paper on the slit lamp examination of the lens, aided by various illustrations. A description was first given of what is meant by an optical section, and the appearance of the normal lens as seen in a thin optical section was described. The reasons, illustrated, were given to show, why many of the features thus seen—such as discontinuity zones—are not visible unless the optical section is very thin. He stated that the newer type of Zeiss apparatus are fitted with an achromatic focusing lens, the use of which appeared not hitherto to have been general in this country. It is essential to use this lens in order to obtain sufficiently thin and clean optical sections.

He emphasized that the relucency of a region affords no indication of its capacity to obstruct the passage of light; and that the curved discontinuity lines in the optical section in no sense represent regions of increased opacity. They are not revealed by retroillumination with the slit lamp, or by retinoscopic illumination against the red fundus reflex. In physics the term opacity is defined on the basis of transmitted, and not of reflected light. Under such conditions the opacity of a medium, acting as a light filter, is given by the ratio of the intensity of the incident light to the intensity of the light transmitted. It is not possible to judge of the opaqueness of any part of a translucent medium, by its effect on light which is incident upon it from the same side as that on which the observer is situated. The medical student who for the first time sees the lens of an old person, readily visible by oblique focal illumination, and on this evidence diagnoses cataract in a lens which is clear to retinoscopy, is falling into the error of judging opacity by direct instead of transmitted light.

An ideally transparent medium has unit opacity of which the density, the logarithm of the opacity, is zero. Because in the direct illumination of the optical section certain features may appear vividly white, i. e., very visible, very relucent, it is inaccurate to say that they are very "dense." It is desirable that ophthalmologists should now come to some conventional decision, as to the nature of the changes to which in future the term "cataract" shall be applied—if the use of a term of such unhappy omen and so much feared by patients, must be retained in ophthalmic practice. It would be a start in one direction if we refrained from using the word "cataract" in respect to features which scientifically are not opacities, i. e., features whose optical density borders on zero, so that they do not demonstrably obstruct light.

The principles of the growth of the human lens and the formation of its suture lines, were referred to in connection with the increased depth of the disjunction lines, which earlier in life were more nearly subcapsular in situation.

The conditions of faint capsular iridescence and minute punctate and linear cortical markings, which, as Vogt has pointed out, in some instances occupy a borderline position between the physiologic and pathologic were then described. It was pointed out that iridescence is essentially a phenomenon seen only when observation is made along the axis of specular reflection. Incidentally, inaccurate focusing of the illumination, so that the reflection surface under observation is illuminated by the extrafocal and chromatic region of the light bundle, may create an apparent faint iridescence which is merely instrumental in origin. The phenomenon of iridescence, when it is pronounced and truly pathologic, has been suggested as tending to discriminate the type of cataract which is a consequence of other ocular changes, from that which is essentially senile in origin. Such a means of discrimination is unreliable.

Pathologic conditions of the lens were then described, under the classification Traumatic, Development (embryonic and infantile), Retrogressive (senile) and Complicating.

A description was given of a variety of traumatic lens changes, which could be detected and located only by means of the slit lamp in the direct illumination of the thin optical section. Originating in a subcapsular situation this

change becomes deeper with the growth of the lens—evidenced as an accessory relucency zone—its situation being related chronologically to the origin of the normal discontinuity zones.

*Discussion.* Dr. William H. Wilmer, of Washington, cited a number of cases in which the use of the slit lamp had been of an aid to him in clinical diagnosis. These cases included a case of facial palsy, in which the poorly nourished character of the cornea could be detected by changes in the corneal epithelium. A case of mild glaucoma, where the diagnosis was made by the bedewing of the cornea and the pigment granules in the anterior chamber, seen with the slit lamp. Dr. Wilmer spoke of a case which had suffered repeated attacks of iridocyclitis, and in which areas of atrophy appeared in the iris. He told also of a case of a very old person who had opacities in the posterior cortex of the lens for over fourteen years, without progress; but recently there has been a thrombosis of the central retinal vein in one eye, with hemorrhages in the retina and vitreous. Following this, changes took place in the lens, giving the appearance of cholesterin crystals in the posterior cortex.

Finally Dr. Wilmer described the appearance of a case of siderosis bulbi, as seen with the slit lamp.

Jonas S. Friedenwald, Editor.

## THE NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Monday, February 16. 1925.

Chairman, Dr. Robert Sullivan.

### Supposed Eye Injury.

Dr. E. B. Cayce showed E. D. S., a white male 30 years of age, a miner by occupation. This patient was seen first by him on January 28, 1925. He gave a history of having been struck on the back of the head by a coal machine jack, and the following day about noon he found the vision of the right eye decreasing.

Examination showed the vision of the right eye to be perception of hand movements and the vision of the left eye to be 6/10 with correction. The pupils reacted normally to light. The right eye was proptosed slightly outwards and showed external strabismus. The fundus showed a detached retina in the macular region and a detachment in the lower part of the fundus, both connected by a fibrous band of white color.

The question for discussion is whether this is a congenital condition or whether it is a recent condition due to the injury which was so slight that he did not report it at the time.

*Discussion.* Dr. W. G. Kennon. I do not believe the injury had anything to do with this eye condition. I believe it is a congenital persistent hyaloid artery. It has a rather peculiar appearance. I think you can catch a view of the opaque material which has practically attached itself to the posterior surface of the lens.

Dr. Robert Sullivan. Dr. Robert Warner and I secured a little additional history from this patient. He states that he has been a railroad man and had an examination by the railroad some two or three years ago, at which time he claims that the vision of each eye was normal. If his statement is correct, this would argue against this being a congenital condition.

### Melanosarcoma of Eye.

Dr. E. B. Cayce reported the following case: Mrs. C. E. M., a white female, 51 years of age, who first consulted him July 16, 1924, stating that she had noticed general failure of the vision for the past year.

Examination showed the vision of the right eye to be perception of light and the tension 42 mm. There was opacity of the lens, rather advanced. The vision of the left eye was 3/10 and the tension was 80 mm. The lens showed beginning opacity, especially below. Pilocarpin muriate and dionin were prescribed. October 22 he did a broad iridectomy on the right eye and about three weeks later delivered the right lens. There was some material, taken to be cortical material, left in the eye. This, however, proved to

be a growth. On January 31, 1925, the right eye was enucleated and a pathologic examination showed melanosarcoma.

Dr. Cayce quoted Dr. G. C. Savage who says that "in forty-seven years' experience, with one hundred or more cases of melanosarcoma, he has never but once seen a recurrence of the growth. This was one in which the sclera had been invaded. Where it was entirely intraocular, he has never seen a recurrence."

**Gun Shot Wound of Eyes.**

Dr. Herschel Ezell exhibited the following case: Mr. R. L. E., a white male, 28 years of age, who consulted him January 26, 1925. The patient gave a history of having been shot in the face and eyes two weeks previously. He was prostrated, bled profusely, and could not see. He was taken to the Nashville General Hospital where he was X-rayed, treated, and remained for about one week. He states that no operation was performed while there. The vision was improved slightly but he could not see sufficiently to go alone when leaving the hospital.

When examined there were a dozen or more wounds on the face, eyelids, etc. The pupils were widely dilated, presumably from atropin which the patient had been using. There was one wound in the right sclera about a quarter of an inch below and a quarter of an inch to the right of the cornea, and the left was very red and inflamed. There were several small nodules in the lids which were apparently due to shot. External examination revealed that the left eye was similar to the right, except that no injury of the ball itself was found. It must be remembered, however, that any wound of the balls had had time to heal before he saw the patient.

Right eye: Vision 20/200. No improvement by glasses. The fundus could not be seen due to vitreous opacities.

Left eye: Vision 20/200—. Ptosis upper lid. Fundus clear and a small hemorrhagic area could be seen in the retina to the nasal side near the equator.

The patient was referred for X-ray and Sweet's localization. These showed many shot around both eyes. There is one shot within the right eye near the corneoscleral junction and one back of the ball close to the optic nerve. There were two shot in the left eye, one anteriorly and one posteriorly.

It was thought best not to attempt the removal of any shot as this would necessitate too much surgery and expose the eye unnecessarily to infection. The patient was therefore put upon argyrol and advised to use hot applications.

February 16, 1925, the patient returned. Eyes much clearer.

Right eye: V., 20/100+. Left eye: V., 20/200. Both pupils widely dilated altho no atropin had been used since January 25th. The ptosis of the left upper lid has practically disappeared. The fundus of the right eye can be seen but is blurred. There are several large stringy vitreous opacities. The optic disc is unusually white and apparently atrophic.

Left fundus. Media clear. No vitreous opacities. Optic disc white. There is one large triangular hemorrhagic area extending from optic disc fan shaped upwards and outwards. This area shows three small black spots near the disc. Above and to the nasal side is a large area of organized exudate, interspersed with small hemorrhagic areas. This is a beginning proliferating retinitis. Near the equator above and to the nasal side is a dark pigmented spot, which corresponds to the location on the X-ray of shot No. 2. The outer lower part of the fundus is free of hemorrhage but is somewhat hazy.

Glasses do not improve the vision of either eye. Subconjunctival injections were not used as the eyes were as much inflamed in the first visit as if they had been used. He is still of the opinion that no operation should be undertaken for the removal of the shot or otherwise and feels that the best results will be obtained by quieting the eye by local applications, eliminants, etc.

*Discussion.* Dr. Robert J Warner: I see no evidence of intraocular for-

eign body in either eye. I do not say that there has not been a perforating injury, but regardless of the X-ray reports I do not believe there is now any intraocular foreign body in either eye.

DR. LESLIE BRYAN: Dr Cayce and I saw this patient at the City Hospital. He had considerable swelling about the face and lids so that the lids could scarcely be separated. We could, however, see quite a good deal of conjunctival chemosis and edema. After treatment for 24 to 48 hours we were able to see the pupils but could not get no intraocular picture whatever. One eye was apparently worse than the other and seemed to be filled with hemorrhage. The other eye showed a slight reflex but nothing could be made out. X-rays were made which showed pictures similar to the present X-rays. The patient had no treatment other than atropin, silvol, etc. We made no attempt at operative procedure because we had no idea where to go or what to look for. The patient remained in the hospital for a few days and then signed out and we have not seen him since.

DR. W. G. KENNON: In this case I would advise what some one has termed "scientific neglect," as in all probability the foreign bodies could not be removed without destruction of the eye.

DR. EZELL (closing): Replying to Dr. Warner I would say that while I cannot prove that shot are present in the eye, on the other hand I am not positive that they are not present. In the left eye to the temporal side there is a good deal of organized exudate behind which the shot could be buried and could not be seen, and that is the area in which the Sweet's localizer has located it.

**Intraocular Tumor (?)**

DRS. W. G. KENNON and ROBERT J. WARNER presented N. B., a colored female, 58 years of age, who consulted them February 16, 1925, complaining of poor vision in the left eye, first noticed about August, 1924. She stated that at times she had pain in the left eye, this pain lasting for a few days.

Examination showed the right eye normal with vision of 20/20. Left eye: V.=P. L. External examination normal. Pupillary reaction normal. By ophthalmoscopic examination nothing can be seen in the fundus, not even the reflex. With oblique illumination a small red streak can be seen in the upper temporal field, this waving slightly with the movement of the eye. By transillumination the lower nasal quadrant is dark, the rest of the eye is normal. Tension 25 mm. by Schiötz tonometer.

The question of diagnosis here is between a detachment of the retina, massive intraocular hemorrhage, or intraocular tumor. Usually in intraocular hemorrhage one gets a fairly good view of the hemorrhage. Where there is detachment of the retina you can see fairly well by oblique illumination, and where you have tumor you are usually able to get a view in some meridian by some type of illumination. But looking at this eye is just like looking at a blank wall. We would appreciate any suggestions in the way of diagnosis.

*Discussion.* DR. HERSCHEL EZELL: While I do not mean to advise enucleation for the sake of diagnosis, yet I believe the only way to make a diagnosis in this case would be after enucleation.

DR. ROBERT SULLIVAN: I believe this is intraocular tumor because nothing else would give the same picture as this does by transillumination, and also because of the slight increase in tension. Normal tension varies between 15 and 25 mm., and for this patient 15 or 20 mm. may be the normal, just as in the case of one whose normal temperature is 96, a temperature of 98 degrees would mean two degrees of fever.

*Note*: This eye was subsequently enucleated and pathologic examination showed total detachment of the retina, and hemorrhage so fresh that it probably occurred at the time of operation. No evidence of tumor.

FRED E. HASTY,
Secretary and Editor.

## MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

FEBRUARY 13, 1925.

DR. J. S. MACNIE, PRESIDENT.

### Ring Abscess of the Cornea.

DR. JOHN ROBINSON (Duluth) presented the case of F. W., farmer, age 54, who was preparing to blast a stump with picric acid when some of the powder blew into his left eye. There was some irritation at the time but no pain until 9 days later. He was first seen on the 10th day. The left cornea showed then a deep annular infiltration, 2 mm. in width, of yellowish grey color, running concentrically with the limbus, and separated from it by two or three millimeters of fairly clear corneal tissue. Under the loupe the epithelium showed no defect; there was also no staining with fluorescein. There was a certain amount of interstitial haze in the central area, but thru this it could be easily seen that the pupil was round and slightly dilated. Anterior chamber fairly deep; tension normal; only slight pericorneal injection. Conjunctiva injected, but no secretion. There was a very small hypopyon. This disappeared promptly under the use of heat, rest and atropin. The pain was controlled by these measures.

The infiltration maintained its ring shape for nearly two weeks, and then gradually spread thru the central area, involving the middle and deeper layers. There were quiet intervals of a day or two during which there was little pain; again for some hours the suffering was severe.

The eye was removed at the end of five weeks. Examination of the fresh specimen showed a necrotic like process confined to the middle and posterior corneal layers. The aqueous was cloudy; Descemet's was partly pushed loose, but there was no pus like accumulation; hemorrhagic engorgement of iris and ciliary body. No suppurative focus within the eye. Posterior segment not involved. Cultures from the anterior chamber showed staphylococcus aureus which produced conjunctivitis and iritis in the rabbit, but not of a very intense type.

During the period of observation the results of the laboratory tests and physical examinations had been negative. The temperature was normal. White cell count 6,000, excepting after milk injections. No suspicion of a suppurative focus was traced which would point to an endogenous origin. There had certainly been no penetrating wound of the bulb. If there was an ulcer, it had healed with rapidity and with few symptoms. In these respects the case is reported as an unusual one. From an examination of the literature it would also seem to border upon the unique in the long duration of the ring formation and in the general lack of extreme severity in the symptoms.

Poisoning by picric acid was suspected; the literature, however, discloses no toxic action in connection with the eye. The reporter desires especially to thank Dr. Alice Hamilton, of Harvard, for her review of her own notes along this line.

Picric acid sprinkled in the rabbit's eye produced only a moderate degree of conjunctival injection.

### Traumatic Myopia.

DR. JOHN ROBINSON also reported the case of M. J., age 33, machinist, who was struck in the center of the left cornea by an iron pin of the diameter of a small lead pencil and 1 cm. in length. The pin was driven with much force. The examination was made half an hour later.

Corneal center shows abrasion of epithelium 3x4 mm. Pupil semidilated, responds very sluggishly to light, slightly eccentric. No hemorrhage; iris not tremulous; anterior chamber normal depth. Tension rather low; trace of pericorneal injection. Fingers counted at 2 feet, but appeared only as shadows. With the ophthalmoscope there appeared only a greyish-green reflex. No evidence of hemorrhage was noted. The retina was entirely in eclipse. Lenticular streaks or dots were absent. The anterior capsule appeared to be intact. As there was some lacrimation, a slightly cloudy corneal surface and mild symptoms of shock, further examination was not persisted in.

The man was hospitalized and kept on his back. Heat and dionin relieved pain which first appeared some hours later. In the ward of a general hospital with the patient in bed, the ophthalmoscope was again used at the end of 24 hours, and again there was no result but a greenish reflex. The general appearance of the eye was unchanged.

At the end of 3½ days the vision had improved to fingers at 10 feet. A distorted view of the fundus was now obtained with —10. Obviously there was much astigmatism but opacities were not noted in the lens. Patient still on his back.

At the end of 4½ days in an examination made in the dark-room, there appeared to be a complete traumatic cycloplegia with the pupil partly dilated. (This condition was, as usual, permanent.) No lenticular opacities were detected. Retinoscopy gave —3.50 sph. with —4.00 cyl. axis 105°. A most striking astigmatic band crossed the pupil obliquely. The vision was 10/200; with the dark-room finding. 20/50—, the best then obtainable. This myopic astigmatism then continued to recede. At the end of 10 days the correction was —0.75 sph, with the same cylinder as before, but the axis changed to 90°. Four weeks after the injury the myopia had gone, but there was a +1.50 cyl. axis 180° added to a low hyperopia. This condition hung on for a month, when finally it too disappeared, leaving an apparently normal error of 2 D. hyperopia. About this time there was first discovered a few grey lines radiating up and inward, apparently deep in the posterior cortex. At the end of a year nothing more of a cataract had developed.

The ophthalmometer showed no corneal astigmatism. The vision ultimately was 20/30 with the normal hyperopia corrected.

The case is reported as one of unusually high traumatic myopia and astigmatism. This condition is usually ascribed to ciliary spasm or relaxation of the zonula. In this instance the obscuration of the fundus, with the subsequent appearance of the few lenticular opacities, suggests an actual swelling of the lens fibers from direct concussion as, at least, a contributing cause of the myopia which apparently reached or exceeded 8 or 9 diopters on the 2nd or 3rd day.

**Dystrophy of the Corneal Epithelium.**

Dr. John Robinson reported a case of a rugged Polish laborer presenting himself July 21st, with a complaint of blurring and watering of the left eye. No injury was mentioned; the man's English was limited and a definite history was not obtained; the trouble had been noticed for several days.

Extending downward and slightly outward from the area of the pupil was a superficial opacity about 3x5 mm. Under the loupe the epithelium, which was chiefly involved, was raised and roughened, and strongly suggested a drying up herpes. There were some fine grey dots on or slightly beneath Bowman's membrane. Fluorescein stained faintly yet quite distinctly. There was some conjunctival injection but none of the pericorneal type —no distinct inflammation. Tension normal.

My impression at the time was that the lesion was not an acute one. The patient came from a neighboring town and was referred to his local hospital to which he had access by an industrial arrangement. The tentative diagnosis was corneal erosion.

He returned August 13th, now claiming that he got dirt or cinders in the left eye two days before I first saw him, i. e., on July 19th. The appearance of the eye was unchanged. No active inflammation, some redness of the palpebral conjunctiva, but little injection of the bulb. Half of the opacity still stained faintly with fluorescein. Under the Zeiss loupe the epithelium had the appearance of breaking-up river-ice, uneven, pushed up in places, here a cake of grey and there a spot still translucent. While there had been a suggestion of herpes three weeks before, now there was entirely no sign of blebs. To the unaided eye the opacity was circumscribed, but under magnification grey dots and lines extended beyond what at first appeared to be a line of demarcation. There was little discomfort. The condition re-

mained unchanged for four months. Treatment had little influence. Moderate photophobia was relieved by a cover, but otherwise the eye acted quite as well when uncovered. The lesion never stained over entirely with fluorescein (or, if so, very faintly) but always there was some portion which took the stain, sometimes in the middle, sometimes below.

During December he had had a "cold" (during an influenza epidemic) and he now returned with the upper third of the opacity ulcerated, and, for the first time, symptoms of iritis showed themselves. This complication, however, was quite promptly relieved by rest, heat and atropin, with one application of iodin to the small crescent of infiltration. In 10 days this infection had passed and by the 1st of January the same lingering opacity again held the stage, roughened, slightly elevated, and cloudy epithelium; this can be easily brushed off and greyish dots then show on Bowman's membrane. Vision: fingers at 8 feet. Seven and a half months after date of first examination the opacity has become rather more dense, but the general character is the same: superficial, grey, 5x5 mm., stains faintly, sharply circumscribed above, fading away into clear tissue below; under loupe epithelium faceted, but no distinct blebs. All else appears normal. The laboratory tests and general physical examination, all carried out in some detail, have given no clue. Sugar has never been found in the urine. Tolerance tests have been made. The blood sugar varies from 128 to 145 mg. per 100 cc. No source of infection has been discovered. Glaucoma has been excluded.

The cardinal signs of dystrophia epithelialis corneae (Fuchs) are loss of tactile sensibility and a certain bleb formation involving the epithelium. In this case the corneal sensibility seems to be lessened in the affected eye, but this is noted after several months of various kinds of treatment; and again, *the fellow eye has quite normal corneal sensibility.* Whatever suspicion there was of bleb formation occurred only early in this case.

The question of injury cannot be altogether ruled out, altho the claim is not well sustained. The employers denied liability; the fellow workmen saw nothing of the accident; the patient himself omitted to mention an injury when first examined.

The slit lamp study was made by Dr. Richard Smith of Superior, who also called attention to what he believed to be a lessening of the corneal sensibility.

*Discussion* (of 3 preceding cases). Dr. John Fulton, in connnection with Dr. Robinson's case of ring abscess, called the attention of the Academy to a case of Mooren's ulcer which Dr. W. W. Lewis reported at the last meeting. He said these two forms of ulcer resemble each other inasmuch as both of them were rebellious to all forms of treatment, usually demanding enucleation. Resistance to disease is usually absent in individual attack by this form of corneal trouble disease. For this reason the foreign protein treatment does not aid us. This, however, should not discourage us, but rather should increase our energy in attempting to work out new remedies for the benefit of this most obstinate form of corneal ulceration.

Dr. W. W. Lewis, in discussing Dr. Robinson's case of traumatic myopia, stated that he had had a case which caused quite a little trouble in settling. The man had a fracture of the rim of the orbit and when the compensation was being considered, the uninjured side had normal vision. The injured side had 1/10 vision and myopia, which corrected gave 10/10 vision. Patient was kept under observation for some time. That myopia gradually diminished from approximately 4 diopters to 7/10 vision without a glass and 10/10 with small minus correction. At first it was difficult to account for, but it may be accounted for in this way. The fracture of the rim of the orbit resulted in considerable inflammatory exudate in the upper part of the orbit and pressed on the upper part of the eyeball, causing axial myopia. As this exudate became absorbed the pressure was less on the eye, with almost a total reduction of myopia in the end.

Dr. Wm. Murray asked if there was any pain in this case.

Dr. Robinson stated that distinct

pain was absent save only during the intercurrent infection in the month of December.

Dr. Murray said it occurred to him that the absence of any marked diminution in sensitiveness of the cornea, which is a prominent sign of dystrophy, might make this diagnosis somewhat questionable.

Dr. Berrisford asked if there had been any pain around the eye.

Dr. Robinson replied that such had not been complained of. He said he assumed that Dr. Berrisford had the neuralgic pain of herpes in mind, and that he himself had suspected this condition in the beginning, but he thought that herpes could be properly excluded. Pain and other evidences of active irritation had, he believed, been absent in all of Fuchs' cases as originally reported in Graefe's Archives.

**Accommodation After Cataract Extraction.**

Dr. Arthur E. Smith (Minneapolis) presented a case of accommodation remaining after cataract extraction, and commented on the unsatisfactory status of our present knowledge of the physiology of the function of accommodation.

The patient, P. G., aged 59, had sustained a severe laceration of the right eye 28 years ago, the eye having been enucleated the following day. Never conscious of any trouble with the left eye until he was 50, when he commenced to wear glasses for reading. No pain or inflammation in the eye at any time but the vision has gradually failed until he is unable to read and has difficulty in making his way around alone. Vision: fingers at 2½ meters.

February 5, 1924—preliminary capsulotomy.

February 6—extraction of cataract (simple extraction).

March 18—Refraction—Plus 10 sph. ⊃ plus 1.75 cyl. axis 160. V=20/20.

This correction was prescribed and he was told that it would be necessary for him to have a separate pair of glasses for close work. He reported a short time later that he was able to read the newpaper comfortably with his distance correction and on testing was able to read Jaeger No. 1 at 12 to 16 inches.

*Discussion.* Dr. John Fulton stated that Dr. Zentmayer has given very careful study to this subject and attempted to point out why some patients seem to retain accommodation after the lens has been extracted. It is impossible, Dr. Fulton said, to imagine that any accommodation remains, and yet we do meet with cases who use the same lens for near and distant vision, having normal vision for both. Dr. Zentmayer attributes this to the bulging of the anterior layer of the vitreous or to contraction of the lid fissure or by patients sliding the glasses down the nose increasing their power, and thus obtain a limited accommodation effect.

Dr. Macnie stated that some years ago while with Dr. Todd, they had a patient who could accommodate after cataract extraction. Upon observing this woman they found that she would get her apparent accommodation by squinting the eyelids. Dr. Macnie anesthetised the eye with holocain and by using a speculum (thus preventing her squinting her lids) she could not accommodate. Patients, by tilting the thick +10 (or thereabouts) lens, can increase the refractive power of the lens 3 or 4 diopters and read with them. Also by pushing the spectacles forward from the eyes they sometimes simulate accommodation.

Dr. Smith stated in reply that he had taken this into consideration, but the position of the lens before the eye was not changed, the patient did not squeeze the lids, and the pupil was rather large with a slight flattening above. In any event, in a case of this kind, a small pupil or a small central opening in the capsule would increase the clearness of vision but would have no influence on the range of accommodation.

**Detachment of Retina.**

Dr. E. A. Loomis (Minneapolis) reported the following case: Miss D. P., age 25, telephone operator, came to my office January 23, 1925, with the complaint that a week previous the sight in her left eye had suddenly gone. She continued working until the nurse noticed her eye was red and sent her to me. She complained of no pain but

the eye burned and she was unable to see.

On examination I found only light perception, tension below normal (now slightly above). The retina was forward and there was no fundus reflex. Retina was detached completely, only being held at the papilla and ora serrata. Retina was especially bulging on the temple side.

History was negative except that January 1st, 1925, she claims she had an attack of unconsciousness which resembled epilepsy as near as I can make out. It was the first time this ever occurred. She has large caseous tonsils, otherwise the nose and throat are negative.

Vision in the right eye is 6/6. The main reason I wish to show you this case is to find out the cause of this condition. The most common causes given are: first, myopia; second, trauma; third, idiopathic. I have seen nothing in regard to epilepsy being an etiologic factor, but thought possibly a cerebral congestion or violent strain might have caused it, or possibly a tumor present which might have torn the retina loose. In the second place I hoped the discussion might bring out something as to your experience in handling such cases, i. e., to treat or enucleate.

I refrained from doing anything surgical on account of this being a total detachment and in the presence of low-grade iritis. As to the differential diagnosis between detachment and tumor:

Detachment—Tension is decreased, fields wavy and tortuous, tremulous retina, trans. shows red reflex.

Tumor—Tension is increased, fields are smoother and straighter, firm retina, no folds, trans. shows dark fundus.

We know that the retina is held against the choroid by vitreous pressure. If there is negative pressure of the vitreous as choroiditis exudativa, or loss of vitreous following operation, the choroid shrinks and pulls away the retina. The same thing happens in positive pressure where there is a hemorrhage behind the retina, or a tumor.

The treatment used was absolute rest in bed on the back; diaphoretic and general treatment with pressure in the hope of absorbing the serous fluid.

Verhoeff gives a method of scleral puncture or trephine opposite the largest amount of detachment following with electric cautery punctures. Perlmann claims that blunt concussion on the eye is never capable of producing detachment in a healthy eye. In treating, his method is to evacuate the subretinal fluid and apply pressure bandages. He claims to have treated 35 cases of partial detachments, 22 men and 13 women. He thinks 10 of them were cured. They varied in age from 16 to 73 years and had existed from 3 weeks to over a year before being treated.

Should one try treatment here, or enucleate the eye?

*Discussion.* Dr. Berrisford said that to him the case was one of complete detachment of the retina. If Dr. Loomis feels that a growth is contained within the eye, there remains nothing to do but to remove the eye. If he considers treating the condition he would better abandon the thought for his efforts will meet with failure.

**Trachoma.**

Dr. J. A. Pratt (Minneapolis) reported two cases of trachoma. He stated that owing to Dr. Fulton's paper at the last meeting of the Academy, he thought it would be interesting to see two cases of trachoma that have been treated with the boric acid treatment.

Case 1. Miss G., was first seen Oct. 22, 1921, after she had been treated elsewhere for six months for an obstinate conjunctivitis. She was treated with boric acid massage and expression of isolated trachoma bodies. The case shows the normal condition following this line of treatment.

Case 2. Mrs. F., contracted trachoma at least 30 years ago; she has been treated by most every method, particularly the blue stone, witnessed by the many adhesions. She was brought to the office June 14, 1924, being unable to travel alone or perform her houshold duties by reason of the

corneal pannus. She was treated by boric acid massage on June 14, 19, 26, and July 2, 9, 16, 31, August 31, October 1, November 3, December 11, and January 29. After the third treatment she came to the office alone and now attends to her regular duties.

*Discussion.* DR. PRATT stated—in response to an inquiry about the boric acid treatment—that this is just a simple mechanical massage. He first thoroly cocainizes the eye, then, making a hard swab on the end of an applicator, dips it into distilled water, then boric acid powder, and massages the lid. The irritation brought on seems to promote absorption and healing. Dr. Pratt said he had not seen a case that did not respond to this treatment. He generally treats a case about every 5 days, or once a week, and continues until the eye is well.

He stated that they are now using a solution of 10% copper sulphat in glycerine, and have the patients use a drop to a teaspoonful of boiled water at first, but when they first started, they used only the first line of treatment.

**Congenital Anomalies of the Eye.**

DR. VIRGIL SCHWARTZ (Minneapolis) presented the case of Mr. A. D. H., aged 56, married, was admitted to the Minneapolis General Hospital on January 27, 1925, for paranoia. Past history is negative. For the past six years he has been a riveter in the boiler room of the Milwaukee railroad shops, swinging a heavy hammer upon the rivets. He states that he has had practically no vision in his left eye for many years, and that the vision of his right eye is failing. He further states that his father and the latter's brother had an eye involvement somewhat like his own; in fact he asserts that eye trouble had been hereditary among the males in his family for several generations. His grandfather's eyes were clear, he says, but his great-grandfather's were also affected.

The patient exhibits a number of congenital ocular anomalies. The right eye shows a coloboma of the iris and a large coloboma of the choroid; there may also be a coloboma of the ciliary body; about this we cannot say definitely. There is also a partially developed cataract in his right lens. It seems fortunate that the nature of his work has required him to look down almost constantly, for the upper part of his retina and choroid are intact; the coloboma, however, eliminates the greater part of his upper visual fields. The optic disc itself seems flattened, if not actually invaginated, inferiorly.

The colobomata, of course, are easily traceable to a failure or incompleteness of fusion of the hyaloid fissure, or the fetal optic cleft, this being always present on the inferior surface of the optic vesicle which is pushed out on each side from the fetal forebrain. If the failure to close does not extend posteriorly, the coloboma will involve only the uveal tract; if only the most anterior portion of the cup shows a deficiency, only the iris may be involved. If this outermost portion is spanned by only a few fibers instead of by a complete closure, an accessory pupil, below the normal pupil, will be formed. Furthermore, if the fissure should shift slightly to one side instead of being in the center of the optic stalk below, an eccentric or ectopic pupil results. This patient's left eye clearly illustrates both these latter conditions.

The cataract in the right eye is only partial, but that in the left is much farther advanced. Their etiology is hard to explain. Fetal ocular inflammation has often been advanced as a theory, while Fuchs believes that a retinal detachment, from adhesion to the margins of the coloboma, may here be present, in which case the lens would suffer thru a change in its nutrition or thru permeation with inflammatory products.

*Discussion.* DR. JOHN BROWN said he believed Dr. Schwartz made a statement that these congenital anomalies, where the fissure is being formed, are due to some defect in closure of the fissure. Dr. Brown was wondering in his own mind how congenital coloboma of the upper segment could be accounted for in that way. He knew of two cases—one, perfectly symmetric from above. In fact he believed there must be more than one way in which these colobomata of the iris could be accounted for.

Dr. Wm. Murray stated that the case of coloboma of the iris and corectopia is interesting to him because he saw this same case about 12 years ago at the City Hospital. As he recalled the man's history, it interested him very much at that time as there was quite a definite history of the same congenital defect being present in 4 or 5 generations preceding.

Dr. Schwartz asked Dr. Brown if the coloboma in his case was in the iris alone.

Dr. Brown stated that it was in the iris alone, and absolutely above.

Dr. Schwartz said that we know that the choroidal fissure at times may not be exactly below, but at one side, in which case a deficient closure there would lead to a coloboma at one side, either the right or left.

In regard to Dr. Murray's statement that this patient gives a history of involvement in several generations, Dr. Schwartz said he had questioned the patient very closely about this and the patient said that his father, his father's brother, and his great-grandfather had an eye condition somewhat like his own. His father's ocular involvement, however, did not prevent him from serving with distinction in the army. With regard to his ancestry farther back than the 3rd generation, the patient said he had no accurate information.

**Melanosarcoma of the Choroid.**

Dr. Virgil Schwartz also presented the case of L. R., a young man, aged 20, white, unmarried. He was admitted to the General Hospital January 9, 1925, with severe pain in the right eye of three weeks' duration. Vision in this eye had been impaired for a long time he said, but about a year ago it became rapidly worse so that for a number of months—almost a year—this eye had been functionless. Aside from the following, his personal and family history were negative. Seven years ago he was struck in this eye with a swiftly-thrown baseball, and had considerable trouble in the eye afterward, tho he had some vision, he says. About a week before admission, the eye became painful and reddened. When seen by us he had marked ciliary and conjunctival injection and the pupil was dilated. Purkinje-Sanson images were present. The eye felt very hard, and the McLean tonometer, which registers a normal tension as being anywhere between 25 and 40, showed a tension in the good eye of 35, while in the affected eye the tension was 120; in other words, an absolute glaucoma existed.

A very disturbing condition was the fact that the fundus was not to be seen. The lens was subluxated, its upper margin being faintly visible, but the interior of the eye was not to be made out.

Because of the clear history of traumatism, it was thought that there might be an old, with possibly a fresh intraocular hemorrhage, despite the fact that the old clot, which would have organized long ago, would probably have produced a retinal detachment and so, usually, a diminished tension. However, a recent hemorrhage, or a transudate, might easily elevate this again. The possibility of intraocular tumor was also suggested, but under the circumstances this could not be investigated. Transillumination was not conclusive. At any rate, the patient was suffering greatly so an iridectomy was done, after having first made a subconjunctival injection of adrenalin as recently recommended, unfortunately without decreasing the tension appreciably in this case. The patient felt better for a few days, the tension having dropped to about 45; but soon this began to rise again and the pain reappeared just as before. More than that, some discomfort and lacrimation appeared in the left, or good eye, so that it was determined to enucleate at once. This was done and on section of the eyeball by Dr. Camp, there was found a melanotic sarcoma of the choroid, somewhat less than a centimeter in diameter and rather near the equator. There was a complete detachment of the retina, being pushed up hard against the iris and lens. Beneath it, and filling the greater part of the eyeball, was a mass of serum, the vitreous having been absorbed. These findings at once explain our inability to see the interior of the eyeball with the ophthalmoscope. It might be

well to recall that sarcoma of the choroid at this early age is very rare.

*Discussion.* Dr. Fred Pratt said that this tumor case gives a definite history of injury several years ago. When he came in he had a dislocated lens, a deep inflammation, dilated pupil and a hard eyeball. It was quite easy to say it was secondary glaucoma. We had hoped an iridectomy would reduce the tension; but the tension stayed up, and increased pain with photophobia in the other eye, decided enucleation. The tumor was only found on section of the eye. This makes it an interesting case.

Dr. John Macnie stated that perhaps some of the men remembered that several years ago he brought a man up from the University Dispensary who presented a peculiar corrugated appearance of the retina below, which they all were unable to diagnose. One month later that appearance had assumed a rounded distinct outline that apparently did not transilluminate. The unanimous verdict was that that eye should be enucleated for sarcoma. Upon enucleation the growth was found to be a benign cyst of the retina. Of course the patient would eventually have lost the sight in this eye anyway. That case deceived all of those who saw it, as they were unanimous in the opinion that it was a sarcoma.

Walter E. Camp,
Recorder.

## CHICAGO OPHTHALMOLOGICAL SOCIETY.

December 15, 1924.

Dr. J. B. Loring, President.

### Choked Disc.

Dr. S. J. Meyer presented the case of a man who had been referred early in December from the Neurologic Department of Cook County Hospital. He was 38 years of age, a painter and decorator, in good health as a rule. About three months previously, he had noticed lacrimation in both eyes, which lasted about three weeks. One night he drank a quantity of moonshine and he was unconscious for about twenty-four hours. Two days later he noticed a slight dimness of vision, which lasted about two weeks, after which the failure of vision became less marked. A month or so later the vision again began to fail, and at present he had difficulty in seeing at all. Was sent to the neurologic ward as a case of spinal lues. A thoro neurologic examination was negative; reflexes normal; history of chancre twelve years ago; no other serious illness.

When he came under observation of the Eye Department a spinal puncture was made, which showed 40 mm. pressure, 69 lymph cells per cmm. Spinal Wassermann was negative; blood Wassermann 3 plus. Fundus examination revealed a bilateral choked disc, when first examined, of about 4 diopters. There was a question as to whether the condition was due to luetic influence or to alcoholic poisoning. The neurologic staff did not agree with eye department as to exact etiology of the case. The latter thought it probably due to the increased intraspinal pressure, and methyl alcohol poisoning on luetic soil. Fields of vision could not be obtained. Ocular tension was R. 10, L. 12. Vision absent in both eyes.

*Discussion.* Dr. George Suker thought this man had what was called a wet brain, and the outcome of the case would be absolute blindness. The strange thing was that the spinal fluid should be negative and the Wassermann three plus. This was quite often the case, particularly true in cases of moonshine poisoning, when the spinal fluid was taken shortly afterward. Five or six months from now the spinal fluid might be as positive as the Wassermann was at this time.

### Traumatic Paralysis of Depressors of Both Eyes.

Dr. S. J. Meyer. This man stated that on July 2nd, 1923, a fellow worker hit him on the head with a hammer. He was unconscious for a few moments, and when he regained consciousness found that he had double vision. He was unable to move the left side of the face, could not read or do any close work, and everything was blurred and dim. The condition has remained practically the same up to the present time. Previous history prac-

tically negative. There was a bump on the left side of the face in the left temporoparietal region, and a partial paralysis of the left side of the face. The eye findings showed a paralysis of the depressors: he could look straight ahead, but not downward, had to bend forward when he walked downstairs. There was partial loss of accommodation. He could read the first line of the chart with plus 2 sphere, R., plus 3 sphere L. There was paralysis of convergence, the eyes were always straight ahead. Unequal pupils, fixed and rigid. The fields, both central and peripheric, were normal, with blind spots in both eyes normal. Blood and spinal fluid negative; X-ray negative; no line of fracture could be detected. The diagnosis was, lesion involving the third, fourth and seventh nerves; the exact location probably the nucleus, or possibly higher up.

*Discussion.* Dr. George Suker thought there was paralysis of the fourth nerve associated with paralysis of accommodation, showing conclusively that convergence was impossible without accommodation. There was probably a nuclear hemorrhage, involving the third nerve nucleus, which accounted for the limitation. There was myosis on one side and mydriasis on the other. In his opinion the man would not recover.

**Congenital Bilateral Ptosis.**

Dr. Michael Goldenburg presented two cases which he had operated, one several weeks previously, and one more recently. The procedure in both cases was that of Hess, which he found offered best results. The interest in these cases, other than the cosmetic effect and improvement, was the economic one, one so afflicted being definitely handicapped to meet the competition of life. His opinion was that these cases should be taken care of long before adult life.

**Congenital Coloboma of Iris, Choroid and Optic Nerve.**

Dr. Wm. F. Moncrieff presented a child who had a convergent squint of about five degrees; astigmatic refraction; vision, R., normal; L., 2/10. The colobomata were symmetric in both eyes; each had a little bridge of iris tissue just about even with the limbus. There were white colobomata of the choroid and optic nerve in each eye.

**Burned Lids and Eyeball.**

Dr. Harry Woodruff presented a man who had suffered a burn ten months ago, from falling during an attack of unconsciousness of unknown origin. He fell against a steam radiator and suffered a terrible burn and the loss of the eye; and very extensive cicatrization of both lids resulted. No operative work had been done as yet to correct the ectropion of both eyelids. The patient was shown for the purpose of obtaining the suggestions, of the men who had done plastic work about the lids, as to the best method of procedure.

*Discussion.* Dr. Michael Goldenburg suggested that the case be let alone for about six months or possibly a year, until cicatrization had entirely ceased and all redness had disappeared, before trying to operate. He had operated a case where cicatrization was not complete, and was not satisfied with the result. It had been necessary to operate for the preservation of vision, which frequently was true where the eye was in danger of being lost. Ordinarily he would recommend letting them alone for a time.

Dr. George Suker was reminded of a number of cases he had seen while in military service. He had suggested to Dr. Woodruff that before attempting any plastic work, the skin, particularly on the upper forehead, should be massaged. The procedure he would recommend was the pedunculated flap graft. The Thiersch graft on the forehead was mean, and there was not enough loose skin to bring it together. If the skin was massaged very forcibly with olive oil, four or five times daily, it would be possible to lift it an inch and a half from the forehead.

**Neoplasms of the Eye.***

Dr. Richard Gamble showed a series of slides, illustrating the various changes produced by the more common types of neoplasms affecting the

*Entrance Thesis.

eye. Four cases of choked disc in various stages illustrated the edematous swelling of the disc, the increase in glial cells resulting from low grade irritation from the edema, large caliber of the veins in early cases, and small caliber of the vessels in the late stage, secondary atrophy, retinal hemorrhages, and hydrops of the nerve sheath. In no case was there any cellular infiltration. A case of optic neuritis was shown for comparison, to illustrate the small degree of swelling and the perivascular lymphocytic infiltration, and the exudate thrown off into the vitreous from the nervehead.

Two sections of carcinoma were shown. One affected the limbus; it had penetrated only a short way into the corneal and scleral fibers, altho eight years elapsed between its onset and the exenteration of the orbit. The other case was a metastatic medullary carcinoma arising in the breast. The choroid and the sclera were everywhere infiltrated by the malignant cells, but none were present in the retina.

Several cases of glioma were shown, first the usual type of glioma endophytum, with the entire bulb filled with the tumor tissue to the point of bursting thru the sclera, and the usual amount of necrosis. Another section showed more marked necrosis. The vitreous chamber was filled by a pale pink mass of amorphous material, which had been the original glioma tumor mass, but which had become entirely necrotic. The retina was entirely destroyed by the growth. The only viable glioma cells were the extensions of the growth onto the iris and posterior surface of the cornea, and back thru the optic nerve, the nerve tissue of which was entirely replaced by glioma cells. The patient from whom this specimen was taken died from extension of the growth to the brain.

The marked necrosis of the tumor in this case prompted a review of the literature to discover if any cases of spontaneous cure of glioma by necrosis had been reported. During the last ten years, five such cases have been reported from Central Europe.

(1) Berta Lindenfeldt, in 1915, reported the case of a child four years of age. One eye was enucleated because a large tumor mass had broken thru the globe. It was glioma. The other eye gave a yellow reflex. Later it was removed, but microscopic study showed the bulb filled with connective tissue and bone. There was no retina and no glioma cells. The eye was, however, atrophic.

(2) Purstcher, in 1915, reported two cases in a family in which glioma was prevalent thru several generations. Three members of this family died from glioma. Two members were examined later in life. Both showed an unusual fundus picture—large yellowish white areas in the retina, some flat and atropic, some more elevated and nodular, surrounded in part by pigment. These were interpreted by Purstcher and by Fuchs as recovered glioma. Both cases had vision of fingers at a short distance.

(3) Meller reported two cases, one in 1915, the other in 1922. The first case was quite similar to Purtscher's but the lesions were larger, more nodular and more vascular. Vision was 20/30. His second case was in a child four year of age. One eye was enucleated and glioma was found. In the retina of the other eye there were many yellow nodules, with tortuous vessels running over them. The case was watched for several months and the nodules became larger. Then the patient disappeared from the clinic. Four years later he was seen again, and by this time there were no nodules, only flat chalky white atrophic areas, and the vessels were no longer tortuous.

Several types of sarcoma were exhibited. A very small spindle cell sarcoma was shown. Its origin from the region of the choriorcapillaris was demonstrated. It was about 3 mm. in greatest diameter, but there was a flat, serous detachment of the retina surrounding it, which extended to the macula. This section was shown thru the courtesy of Dr. William H. Wilder, who enucleated the eye. He had made the diagnosis by observing rapid growth in the size of the mass, and by the reduction of vision when the retinal detachment occurred. Several cases of more advanced sarcomas of

the choroid were shown, illustrating their tendency to break thru the retina and proliferate in the vitreous chamber, and to cast off free sarcoma cells into the interior and even anterior parts of the eye. Routes of extrabulbar extension, via the vortex veins, the posterior ciliary vessels and nerves, and optic nerve were shown. In two cases with tumors of the same size in the same location, one showed peripheric anterior synechia with the entire lens and iris system pushed forward, and one did not, indicating that the size of the tumor may not be the only cause of secondary glaucoma. One case of ring sarcoma of the ciliary body showed extension of the growth over the anterior surface of the iris, dipping into the iris crypts.

In a case of retrobulbar sarcoma, several large masses of tumor cells were seen clustering about the optic nerve. In the bulb was seen the beginning of an anterior peripheric synechia, evidently due to pressure on the bulb from behind.

*Discussion.* DR. WILLIAM C. FINNOFF, Denver, Colo., complimented Dr. Gamble on the sections shown, illustrating the various pathologic conditions found in the eyes and the complications that occur with various tumors. He thought they illustrated particularly the need of study of the microscopic sections of the globes by the ophthalmologist. These should be properly fixed at the time of enucleation, and should be studied by an eye pathologist. The average general pathologist was not competent to pass judgment on an eye. In the majority of general hospitals, only a general pathologist was employed. In his opinion, it should be insisted upon that the pathologic material in general hospitals should ultimately be passed on by an eye pathologist. The reports received were frequently wrong, and one was frequently misguided.

For example, sections from the globe of a boy twelve years of age were reported glioma of the retina by a general pathologist. When the sections were secured and examined, it was found to be a sarcoma of the choroid, which had broken thru the lamina vitrea and infiltrated the interior of the globe, but because of the position of the cells the general pathologist had called it a glioma.

Glioma of the brain arises from glial tissue, while retinal tumors originate from retinal cells. Pathologically these cells were different. There was some question at present as to whether all pigmented tumors were of ectodermal origin; this he questioned.

In Denver, there was some difficulty in getting specimens, as there were not so many eye hospitals, but he believed that the general pathologists were beginning to realize that they were not competent to pass upon eye material, and that it should be turned over to an eye pathologist.

## THE MEDICAL SOCIETY OF LONDON (ENGLAND).

MARCH 9, 1925.

DR. EUSTACE M. CALLENDER, PRESIDENT.

### The Fundus Oculi in General Medicine.

MR. ERNEST CLARKE said the service of the ophthalmoscope in medicine had been enormously increased during the last few years, especially since the introduction of the luminous ophthalmoscope, the latter advance having brought the subject of ophthalmoscopy within the practical reach of every medical practitioner. As this latest developed instrument could be brought almost into touch with the cornea in use, the darkened room was not needed, the patient could be examined in any posture, and interference of observation by refractive troubles was almost nil.

It was one thing to see the fundus, quite another to interpret what was seen. One authority stated there were 90 varieties of "normal" fundus, a number which was probably not an exaggeration if included in this was the great variety of vessel distributions. Only by observing many fundi, both normal and in disease, could the practitioner recognize departures from the usual. Failing this experience, he was apt to be alarmed when he saw gross changes, which might not be of clinical significance, such as opaque nerve fibers and colobomata; whereas he

might miss the changes which the ophthalmoscope showed to be present in arteriosclerosis.

In former years it was not easy to obtain accurate fundus pictures for study and comparison; it was important to avoid wearying the patient, and the eye must not be illuminated too long at a time. On the suggestion of Dr. Rayner Batten, Messrs. Hamblin had now set up a studio for this work, and at a few minutes' notice they undertook to take a picture of the back of the eye while the patient was under the mydriatic; they also would take a series of pictures of the same eye, enabling the ophthalmic surgeon to compare the conditions, without having to trust either to his memory or to a hasty sketch and a few words of description. Mr. Clarke said drawings in color were always more useful than those in black and white. Lantern slides of these pictures could also be made, hence it was a great accession for teaching purposes.

Mr. Clarke showed, on the epidiascope, a large series of colored drawings to illustrate his thesis, the illustrations being arranged to illustrate the changes seen in connection with various diseases.

*Discussion.* Dr. F. Parkes Weber said that once when he was examining the fundi of a patient he died, and, continuing his examination, he noticed that the columns of blood in the vessels took on segmentation, i. e., the actual blood was interrupted by small spaces, and these spaces could be moved up and down by exerting pressure on the chest. Later, of course, the cornea became too cloudy and opaque for the observation to be continued.

Dr. Rayner Batten agreed as to the importance of having consecutive drawings in many cases, so as to determine in what direction a case was going. It was difficult to classify fundus pictures according to the diseases, and the difficulty was increased by the reluctance of the medical man sending the case to provide notes of the patient's condition. Such a note was very important, and should in all cases be supplied. Dr. Batten also projected a number of very instructive slides from his series.

Dr. James Collier, a neurologist, said that an ophthalmoscopic examination should be made in every case of illness whatsoever. It was a superficial examination which could be done in a moment, like looking at the tongue or feeling the pulse, and it afforded valuable information. The beginner could and should practice on normal subjects; only in that way could he know when he was in the presence of the abnormal. Every student in his wards was obliged to use the ophthalmoscope on every patient; he reported what he saw, and the speaker criticised his report or confirmed it. He said American physicians had remarked to him that in England more attention was given to the ophthalmoscope than in America.

In many diseases the first symptoms might be ocular; but even more important than these positive findings were negative reports that the fundi were normal. Recurrent headaches, unexplained vomiting, transient blindness, giddiness, and many other symptoms suggested a gross intracranial lesion, and when there was no ophthalmoscopic examination it was impossible to treat the case without having a lurking suspicion that there was a serious organic underlying condition, and that therefore valuable time was being wasted. He had seen difficult diagnostic problems settled by recourse to the ophthalmoscope. Of this he related a number of instances.

Sir William Willcox agreed that if the ophthalmascope were used on every case, much more knowledge would be available on the relationship of changes in the eye to general diseases, and diagnosis would be more accurate. He had recently been much interested in the changes in the fundus oculi from toxemia. Until comparatively recently changes in the fundus were considered to be due to one of three conditions: diabetes, Bright's disease, high blood pressure. But it was very common to find marked changes such as hemorrhages in the fundus in cases having no increased blood pressure and in which the renal function was not disturbed. In such cases he considered it most import-

ant to search for possible channels of infection: teeth, antra, sinuses and so on.

He had seen a number of toxic cases with retinal changes, and he removed the focus, following it by Plombiere treatment, to eradicate secondary intestinal invasion, and then the retinal condition cleared up. Certain drugs appeared to have an affinity for retinal tissue, and among these were atoxyl and quinin. Veronal also seemed to have a lasting effect; he was still seeing cases showing a curious nervous effect of the administration of that drug during the war. He suggested that persons having retinitis showed themselves to be sensitized to the toxin; and when giving a vaccine to such, a commencement should be made with very small doses, a few thousands, instead of the millions usually given. He regarded this sensitisation as akin to that in asthma and angioneurotic edema.

H. Dickinson, Reporter.

## WHY THE TRANSPARENT LAYERS AND MEMBRANES OF THE CORNEA AND LENS ARE THINNER IN THE AXIAL THAN IN THE PERIPHERAL ZONE.

Dr. Leonard Koeppe, Halle, Germany.

This is an abstract of an address delivered before the New England Ophthalmological Society, March 17, 1925.

The fact that all transparent layers and membranes of the living cornea (i. e. of epithelium, of Bowman, of corneal lamellae, of Descemet, of endothelium) and of the anterior and posterior half of the crystalline lens (anterior and posterior capsule, epithelium of the anterior capsule, layers of fibers) are thicker in the peripheral than in the axial zone, is one of the wonders of Nature, and is a necessary correction for the serious optical difficulty which would otherwise exist.

If we make an experiment with a glass cylinder closed at one end with an elastic rubber membrane, and if we fill this container with water, the pressure of the water is directed vertically downward against all parts of the elastic membrane, producing a curvature of the latter. As exact mathematic calculations show, this curvature follows the socalled catenary curve, an arithmetical curve of a higher order. The cornea and the contractable lens substance are fixed to an almost inelastic ring setting (the sclera and the ciliary body respectively), and they are composed of elastic lamellae and membranes. (The two halves of the lens substance may be considered here as a half fluid elastic medium). Then the anterior and posterior surfaces of the cornea and crystalline lens would assume the curvature of the catenary, as a result of vertically diverted pressure, if all the layers of the curves and crystalline lens would have the same thickness in their axial and peripheral zones. Therefore, in the socalled optical zone of four millimeters diameter (Gullstrand), would arise troublesome astigmatism and enormous spherical aberration around the axis.

The analytic calculation shows, furthermore, that the increasing diminution in thickness of all named layers and membranes toward the optical axis causes, in the optical zone of the living eye, satisfactory optical spherical correction of the catenary curvature, in an approximately spherical sense, and for that reason a corrected visual power of our eyes, in all stages of refraction and for all meridians.

Keratoconus shows a too intensive diminution of the axial thickness of the cornea, and it can be considered as a too intensive optical correction of the latter in the optical zone. Megalocornea, on the other hand, has too small a diminution in thickness of the layers and membranes of the cornea in the axial zone, and therefore a too small spherical correction. Megalocornea follows with its curvature more the curvature of the rotation body of a catenary curve.

The whole lecture was well illustrated by blackboard sketches made by the speaker.

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## VISION AND EARNING ABILITY.

Roughly speaking, half of our standard vision may be regarded as surplus; unused in the ordinary occupations of life, but available for emergencies; a provision for loss by accident, or the inevitable deterioration that comes, sooner or later, from senile changes. We can see about as much with one eye as with two. Even for judgment of distance, monocular vision can be developed to serve most of our needs as well as binocular. The loss of one peripheral temporal field can largely be compensated by the habit of using the remaining eye, turning the head, and by mental alertness. The greater acuity of binocular vision over monocular vision, only becomes perceptible when illumination has been greatly reduced.

With vision only 6/10 of the standard vision, half of the 12/10 possessed by many people, reading is possible, even of the finest type that can be cast in type metal; sewing, even fine sewing, can be carried on; fine mechanical work, even watchmaking can be continued, without recourse to unusual optical assistance. Marksmen, plainsmen, lookouts at sea, may all continue to render accustomed service. Nor does such reduction of vision necessarily entail notably greater danger of eyestrain.

The diminishing of retinal contrast, diminishes rather than increases retinal fatigue, and the confusing influence of after images. To have one part of the retina adapted for light and the adjoining part adapted for darkness may be a serious cause of annoyance and fatigue, which is greater the closer the differently adapted points are to each other. Retinal diffusion that does not seriously impair perception and interpretation of images, recognition of objects on which attention is fixed, is by no means a serious or unmixed disadvantage.

Full visual acuity is generally desirable, but it is no more essential to industrial efficiency than good education, or harmonious home surroundings. Visual acuity and industrial efficiency can either be expressed in decimals, or other fractions. But visual impairment and industrial impairment are not expressed by the same fraction. They are quite different; and the impression that one is synonymous with the other needs to be corrected. It is still too common among medical witnesses and industrial commissions, and is even made the basis of legal arguments and judicial decisions in the courts. The

rule of reason, lately recognized and formulated, but fundamental to all attempts to secure justice between man and man, commands that we should distinguish between such essentially different things as visual acuity and earning ability.

E. J.

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## OCULIST OR OPTOMETRIST?

Who will prescribe glasses, and who will make them and fit them to the face, will not be finally decided by legislative enactment; no matter how many laws relating to it may be placed on the statute book. The attempt to create a profession by giving its representatives power to say who shall and who shall not render certain service, is doomed to failure. Any attempt to give a legal monopoly of prescribing or fitting of glasses, to those who are not the most competent for that work, may do harm and cause confusion for a time, but such monopoly cannot be permanent. This applies equally to optometrists and to oculists.

The attempt to create a profession of optometry, by passing laws defining it and appointing boards to administer them, has been pushed vigorously by business men who saw in it an opportunity to sell more spectacles and keep up the price for them. It appeals to the self esteem of those who think it desirable to be lifted out of a mechanical trade into a profession; without having to submit to the long, hard educational process, that has made a real profession more highly esteemed than a trade. Laws that permitted those already in the business to continue in it, with only a formal registration and payment of a nominal fee, while making it more difficult for others to get into the business, are likely to receive enthusiastic support from those who are already in; and energetic, optimistic boosters and organizers have found optometric societies as good a field to work as any trade union.

But all this will have little influence on the final result. Those will serve the community who can serve it most efficiently and economically, those who have had the most effective preparation for their work; have given the most time and energy to such preparation, by the best planned course of study. Here is where the optometrists have failed to establish a profession. They have two good schools, with university connections, that give adequate courses in physiologic optics; but how many of the "registered" optometrists of America have taken courses in such schools?

It is often stated that there are not enough qualified oculists to make the needed measurements of refraction for the whole population. This is quite true. But how many towns or cities of 10,000 inhabitants have in them one of the graduates of these good schools of optometry. The majority of schools of optometry are organized on a strictly commercial basis; are more commercial and give less attention to professional ethics and professional ideals, than did the privately owned medical schools of a century ago, to say nothing of the low standard of preliminary education required.

But the crucial question is: what kind of education fits a man or woman to advise people with regard to their eyes? A person wears glasses to get relief from symptoms—pain, headache, congestion, dimness of vision, fatigue, dyspepsia; and a score of others that may arise entirely apart from any need for glasses. Can the most elaborate training in optics, without medical training in pathology and general diagnosis, enable one to discover the cause of the symptoms; and assign to any one of many cooperating causes its relative importance?

People seek glasses for relief from the effects of many diseases, ocular, nasal, cerebral and general; can one who is ignorant of medicine recognize and discriminate among the many possible pathologic conditions, which is present, or which the most important? If the sufferer went each year to the neurologist, the oto-rhinologist, the specialists in renal and cardiovascular disease, the gastrologist, the masseur,

the radiologist and several laboratory workers, he might consult an optometrist, graduated from one of the good schools of optometry, if such a person could be found, with comparative safety. But would such a routine cost less than to consult an oculist, trained in medicine, or a general practitioner who would refer him to an oculist?

The body is a whole, one and indivisible, in pathology and for purposes of diagnosis. It can best be cared for by those who understand this, and have been broadly trained in all that pertains to the body and its health. So that, however they may specialize, they have a good understanding of the limits of their specialty, and can intelligently advise as to what direction relief for any particular symptom or group of symptoms should be sought. The community will get the best service from the profession whose members enter their specialties fitted by common training, and mutual acquaintance and respect, for effective team work, for cooperation that places at the service of every patient the wide resources of modern medical science and art.

E. J.

## BOOK NOTICES.

**Ophthalmometry.** A Practical Textbook for the Practitioner and Advanced Student in Optometry. By **E. Leroy Ryer,** Opt. D., D. O. S., F. A. A. O. Published by the Optical Publishing Co., New York City, 1925. 220 pages, 59 illustrations.

This book is divided into five parts, preceded by a preface. Its raison d'etre may be summed up in a paragraph taken from its preface: "To prove whether or not the cornea is regularly spherical or toroidal in form; to know, if toroidal, the exact dioptric value of each of the two principal meridians; to know that these values are fixed and essentially independent of any ciliary action; to know the amount and the nature of any change that may take place from time to time in the contour of the cornea; to know whether a cornea is symmetric or irregular in curvature; to know, in a word, the precise value of the refraction power of the cornea, the exact amount of corneal astigmia, and the definite location of the axis meridian, is to know that which may prove of inestimable value in the final analysis and ultimate welfare of the case."

Part one treats of the principles common to all forms of ophthalmometers, and part two, three and four describe and illustrate the Universal, the C-I, and the Sutcliffe ophthalmometers, respectively. Part five describes 16 illustrative cases.

Many oculists neglect the use of the ophthalmometer, prescribing upon the basis of data obtained subjectively thru the use of the astigmatic dial, etc., or objectively thru retinoscopy, or a combination of both. It is undoubtedly true that in many cases the ophthalmometric findings must be disregarded, in view of the data obtained from the complete examination. But it is just as true that in a larger number of cases the ophthalmometric findings serve as an index to our final results, and greatly lessen the labor of attaining them. Altho the book was written for optometrists, there is much of value in it for oculists, well written and well illustrated.

C. L.

**Transactions of the Pacific Coast Oto-Ophthalmological Society, Twelfth Annual Meeting, Portland, Oregon, July 10-11-12, 1924.**

This country is so vast and medical, and business centers so far apart in distance and in time, that either national scientific socities have found it necessary to establish local sections, or persons interested in various branches of science have associated themselves in geographic groups. Thus it is with ophthalmology; there being several highly organized and well attended associations.

The Pacific Coast Oto-Ophthalmological Society was founded twelve years ago, and has held annual meetings since that date, in which valuable contributions have been made to eye, ear, nose and throat literature. The volume of transactions for this year

embraces 144 pages, including the list of members.

Of the papers presented in Portland dealing with the eye there were the president's address, Frederick A. Kiehle, on the "Future of Ophthalmology"; the address of Frank E. Burch of the University of Minnesota, "The Influence of High Blood Pressure in Cataract Surgery"; Hans Barkan, "Some Historical Aspects of the Cataract Operation"; Theodore C. Lyster, "Demonstration of a Visual Field Apparatus"; Harry Vanderbilt Wurdemann, "Suturing of the Conjuctiva in the Cataract Operation"; John E. Weeks, "Impressions of Ophthalmologic Activities Abroad"; Hayward G. Thomas, "Glaucoma and the Focal Infections"; Arthur Curtis Jones, "A Case of Orbital Hemangioma Treated with Radium"; Will Otto Bell "Twenty-four Lantern Slides of the Human Fundus"; George W. Swift, "The Cerebroventricular Study."

The discussions were particularly pertinent and in some cases more enlightening than the papers themselves. Most of these have been published either in the AMERICAN JOURNAL OF OPHTHALMOLOGY or in the *Northwest Medicine*.

There were a number of other papers read upon ear, nose, and throat subjects, of which the ones on "A Study of the Eustachian Tube with Special Reference to the Recognition and Preservation of its Delicate Functions in the Ventilation of the Middle Ear", by Frank E. Brown; "The Significance of Bacteremia Following Mastoid Operations", by John H. Harter; "Analysis and Correction of Hearing Defects", by Isaac H. Jones and Vern O. Knudsen, and "The Maxillary Sinus Operation", by Richard W. Perry are significant of progress in these specialties.

H. V. W.

**Transactions of the Section on Ophthalmology of the American Medical Association.** The Seventy-fifth Annual Session, held at Chicago, Illinois, June 9 to 13, 1924. (See also p. 586.)

Of the nineteen papers read before the Section a few stand out: that by the chairman, George S. Derby, on "Standards of Ophthalmology"; "The Toxemias of Pregnancy from an Ophthalmologic Standpoint", by Robert Cartwright Cheney; and "Conclusions Concerning a Sclero-Conjunctival Suture in Cataract Extraction", by Harvey J. Howard. The meetings of this association are always well attended and the discussions productive of progress in ophthalmology. Many of these papers are likewise published in the Journal of the American Medical Association.

H. V. W.

**The Ophthalmic Year Book, Volume XX.** Containing Bibliographies, Digests and Indexes of the Literature of Ophthalmology for the Year 1923. Edited by Edward Jackson and William H. Crisp. Nine illustrations in the text. Published by the Ophthalmic Publishing Company, Chicago, September, 1924.

This review of the literature of Ophthalmology, including the digest of literature and bibliographies, began in 1903. In 1918 it was merged with other publications, but in the year 1923 the original form of an annual volume was resumed. This book gives abstracts of the most prominent articles appearing in ophthalmologic literature as well as a list of all articles published, under appropriate headings. It is primarily a work of reference and, as such, is essential to a well conducted library.

H. V. W.

# ABSTRACT DEPARTMENT

Reprints and journal articles to be abstracted should be sent to Dr. Lawrence T. Post, 520 Metropolitan Building, St. Louis, Mo. Only important papers will be used in this department, others of interest will be noticed in the Ophthalmic Year Book.

**Elschnig, H. H. Ruptures of Descemet's Membrane in Hydrophthalmus and Its Early Perforation in Purulent Keratitis.** Klin. M. f. Augenh., 1924, v. 73, 395.

The clinical history of bilateral congenital hydrophthalmus with intense opacity of the cornea of a child aged 6 weeks, showed that after 4 years the hydrophthalmus had spontaneously healed with subsidence of the opacity of the right cornea. In the left eye sclerotomy had been performed, but from the experience with the right eye, its recovery was probably also spontaneous. A review of the literature is given. The author summarizes, that the well known ribbon shaped rupture of Descemet's membrane of Haab in hydrophthalmus most likely arises very early, some certainly before or at birth. There may be severe corneal opacities which clear up with regeneration of the endothelium. After disappearance of the opacity, the rupture can be easily recognized with loupe and slit lamp. The early perforation of Descemet's membrane in serpent ulcer may in some cases be diagnosed clinically. Generally it is followed by total perforation, but exceptionally, it may remain isolated and the overlying tissue may be preserved as a cicatrix under the regenerated epithelium.

C. Z.

**Franke, C. Blue Sclera, Fragility of the Bones and Otosclerosis.** Klin. M. f. Augenh., 1924, v. 73, p. 119.

Of a family of three sons and two daughters, both daughters had blue scleras but only the older one, aged 14, had sustained fractures. These were of the left leg at the age of 12, of the left humerus at the age of 13, and two months later another fracture of the same humerus below the first. Another case, a girl with blue sclera, aged 10 years, had eleven fractures of arms and legs without special causes. Three younger brothers had blue scleras, but no fractures. There was no otosclerosis.

From the discussion of the literature it appears that heredity is not absolutely certain from the mother's side, while in a series of cases the transmission from the father was certain. Out of 125 individuals with blue scleras, 84 had fractures of the bones. In the second case, the calcium content of the blood of the patient and her father was increased, perhaps due to a hypofunction of the endocrin glands or a hyperfunction of the epithelial bodies.

C. Z.

**Jaeger, E. Sympathetic Ophthalmia.** Klin. M. f. Augenh., 73, Nov.-Dec. 1924, p. 714.

Jaeger presents statistics on 70 cases of sympathetic ophthalmia from the eye clinic of Tübingen with brief clinical histories, with the following summary: Sympathetic ophthalmia after an operative traumatism, especially cataract extraction, is very grave and mostly terminates unfavorably. After simple iridectomy it is rare and not as malignant. Sympathetic ophthalmia chiefly in the form of choroiditis and papilloretinitis, involving essentially the posterior segment, shows a less serious course than iridocyclitis. Eyes affected with sympathetic ophthalmia are very sensitive to later operations, generally reacting with grave relapses, which partly lead to loss of sight. The enucleation of the exciting eye has, even after the outbreak of sympathetic ophthalmia, in the great majority of cases, a favorable influence upon its course. This is the more effectual the sooner it is performed, i. e., the shorter the interval between the beginning of sympathetic ophthalmia and enucleation of the first eye. With great probability the 15th day after enucleation is the last date on which sympathetic ophthalmia of the second eye may occur.

C. Z.

**Schiötz, H. Tonometry.** Brit. J. of Ophth., vol. 9, April, 1925, p. 145.

The author's more recent measurements show that the graphs hitherto used are too low. During the past year he has measured thirty dead eyes in situ and on this basis compiled fresh graphs which are now published in this contribution.

The measurements were made with his old standard tonometer which is always used as the standard for the adjustment of other instruments. Of the thirty eyes, twenty were from individuals dead 8 to 48 hours and ten from one to two hours. In the tables the maximum, minimum and average values are noted. An illustration of the author's improved instrument, three tables and three figures with graphs are presented.

The latest model retains the principle of the older one, a difference being that the 5.5 gm. weight screws on and the other weights are placed directly on the 5.5 gm. weight. The cylinder is now equipped with ball bearing. All tonometers should act alike. This could be accomplished by establishing centers which would cooperate for adjusting these instruments.

D. F. H.

**Schnyder, W. Occurrence and Morphology of Diabetic Cataract.** Klin. M. f. Augenh., 73, Sept.-Oct., 1924, p. 418.

Schnyder describes the development of diabetic cataract in a woman, aged 33, observed with slit lamp. The first notable changes consisted in a strikingly light prominence of the superficial lens fibers. At the same time, subepithelial cloudy opacities appeared under the anterior and posterior capsules, in front, gray, transparent; at the posterior pole, white; not spreading to the cortex. Under the capsule lay spindle and lancet shaped gaps of the superficial layers of fibers. Then the subcapsular opacities cleared up and disappeared and under confluent subcapsular vascular formation, a sudden swelling of the lens set in thru the development of water clefts in radial and concentric directions. The most striking characteristic of this swelling was that the anterior capsule was separated from the fibers by a continuous striation of fluid, in which were globular drops, probably myelin.

C. Z.

**Elder, W. Stewart Duke. Changes in Refraction in Diabetes Mellitus.** Brit. J. of Ophth. vol. 9, April, 1925, p. 167.

The author discusses in considerable detail three of his cases illustrating three types; one, showing refraction varying with blood sugar content, second, showing hypermetropia with sudden decrease of sugar and third, showing myopia with increasing sugar. The contribution contains seven charts and a bibliography of fifty-six. From a study of this literature, forty-five recorded cases have been tabulated. The theories accounting for these changes are discussed under the headings of dynamic, dependent on a neuromuscular mechanism; and static, dependent on a physicochemical mechanism. The writer concludes by suggesting that;

1. In diabetes sudden changes of refraction occur. In the course of the disease with high or increasing sugar, myopia tends to occur; with decreasing sugar, and, therefore, usually just after the onset of energetic treatment, hypermetropia tends to occur.

2. The phenomenon is due to osmotic processes involving the lens, caused by a variation of the molecular concentration of the blood and tissue fluids with the sugar content.

3. In both cases the tendency is to return to normal; in the first, under general diabetic treatment; in the second, on the establishment of metabolic stability. Provided the diabetic element can be successfully dealt with, invariably a good prognosis can be given. The patient can be assured that his eyes will return to normal within a few weeks and that his vision will be unimpaired.

4. In consequence of the general use of insulin, it is anticipated that the rarer condition of hypermetropia will become more common. D. F. H.

**Byers, W. Gordon M. Two Cases of Traumatic Keratitis in the New Born.** Brit. J. of Ophth., vol. 9, April 1925, p. 162.

The observer reports two cases and as a postscript a third, detailing his findings in the cornea with the slit lamp. The first was in a baby aged two days, also observed at aged 6 years, with a band like opacity; by retroillumination a cirrus like opacity. In the second case there was observed, three days after birth, a band like opacity which by retroillumination presented a rupture of Descemet's membrane. The two cases fall in the third class of Thomson and Buchanan's arrangement. The third case (postscript) was observed at 6 years. The opacity was triangular and by retroillumination showed the scar of a ruptured Descemet membrane.

D. F. H.

**Caspar, L. A Tumor of the Hypophysis.** Klin. M. f. Augenh., 73, 1924, p. 172.

A man, aged 55, suffered for some time from headaches and two months ago noticed impairment in the right visual field. There was complete right temporal and partial left temporal hemianopsia for white and colors. The diagnosis of hypophysis tumor was supported by the roentgen picture of enlargement of the sella turcica and diminution of the clinoid processes. The operation with submucous resection of the hard palate and osseous nasal septum, opening of the sphenoid sinuses and chiseling thru the floor of the sella revealed a soft adenoma, which was curetted. The patient left the hospital after two weeks. Gradually a thrombosis of the inferior vena cava (as shown by the autopsy) developed and the patient died from bilateral pyelonephritis. The unusual finding was a hole formation in the chiasm produced by the tensely stretched anterior communications.

The author also gives a brief survey of the eye symptoms of 7 other cases of tumors of the hypophysis.

C. Z.

**Judin, K. S. Intracranial Echinococcus Perforating into Orbit.** Klin. M. f. Augenh., 1924, v. 73, p. 169.

A woman, aged 24, suffered for four years from headache and two years ago became blind in her left eye from atrophy of the optic nerve. Exophthalmus downward and inward by a tumor which could be felt in the upper temporal quadrant, adherent to the eyeball and bone. At the exenteration of the orbit an echinococcus cyst was removed which had penetrated the orbital wall from the cranial cavity. Vomiting and headache persisted for a month with bulging of the remnant in the orbit. An incision thru the skin of the lid evacuated a second echinococcus cyst in the brain substance. Apparently the cyst originated in the frontal lobe. The optic neuritis of the other eye, which seemed to be due to meningitic irritation, disappeared after the second operation. C. Z.

**Mans, R. System of Epithelial Fibers of Cornea.** Klin. M. f. Augenh., 1924, v. 73, p. 289.

Analogous to the epithelial fibers of the skin described by Unna and Frieboes, Mans found by staining after the method of Unna, that the epithelium of the cornea is pervaded by a system of fibers from Bowman's membrane to the surface. It forms around the nuclei, densely interwoven fibers, like a basket (the former cell membrane), and probably passes into Bowman's membrane. In the same fashion, a connective tissue was observed. In inflammations great changes take place in the epithelial fibers, especially after detraction of Bowman's membrane. After recession of the inflammation, an entirely unphysiologic epithelium may develop. If Bowman's membrane is destroyed, the epithelial fibers drift continuously into the subepithelial connective tissue. C. Z.

**Jaensch, P. A. Subconjunctival Injections of Suprarenin.** Klin. M. f. Augenh., 73, Nov.-Dec. 1924, p. 665.

Forty-two cases of glaucoma and eleven other patients were treated with subconjunctival injections of 0.2 cc. suprarenin solution 1:1000. In five

cases of primary glaucoma, not operated upon, the tension decreased from 70 to 50 to 25 or 20, but after a few days showed inclination to increase; in five the injection was without result, and in three doubtful. The suprarenin injections have effectually augmented the number of medicaments for glaucoma. In some cases, not recognized beforehand, they essentially lower the tension, but they cannot supplant operative treatment. In chronic glaucoma their trial is worth while. If successful, operation may be postponed, if not, operation must be resorted to even if tension is temporarily lowered. In combination with pilocarpin and eserin, the reduction of tension after suprarenin lasts about two weeks. Especial advantages are: examination in mydriasis without increased tension, lower tension and thus avoidance of sudden hypotension in eventual operations, which are less painful and less bloody (iridectomy).

C. Z.

# NEWS ITEMS

Personals and items of interest should be sent to Dr. Melville Black, 424 Metropolitan Building, Denver, Colorado. They should be sent in by the 25th of the month. The following gentlemen have consented to supply news from their respective sections: Dr. Edmond E. Blaauw, Buffalo; Dr. H. Alexander Brown, San Francisco; Dr. V. A. Chapman, Milwaukee; Dr. Robert Fagin, Memphis; Dr. M. Feingold, New Orleans; Dr. Wm. F. Hardy, St. Louis; Dr. Geo. F. Keiper, LaFayette, Indiana; Dr. George H. Kress, Los Angeles; Dr. W. H. Lowell, Boston; Dr. Pacheco Luna, Guatemala City, Central America; Dr. Wm. R. Murray, Minneapolis; Dr. G. Oram Ring, Philadelphia; Dr. Chas. P. Small, Chicago; Dr. John E. Virden, New York City; Dr. John O. McReynolds, Dallas, Texas; Dr. Edward F. Parker, Charleston, S. C.; Dr. Joseph L. McCool, Portland, Oregon; Dr. Richard C. Smith, Superior, Wis.; Dr. J. W. Kimberlin, Kansas City, Mo.; Dr. G. McD. Van Poole, Honolulu; Dr. E. B. Cayce, Nashville, Tenn.; Dr. Gaylord C. Hall, Louisville, Ky.; Dr. Edward D. LeCompte, Salt Lake City.

### DEATHS.

Dr. Albert Rémy died at Plombières-les-Dijon, aged 66 years.

Dr. Roy T. Urquhart, Grand Rapids, Michigan, aged 47, died suddenly May 18th.

Dr. Charles R. Lockwood, Kankakee, Illinois, aged 48, died May 23rd of tumor of the brain.

Dr. Lewis G. Woodson, Birmingham, Alabama; professor of diseases of the eye, ear, nose and throat, Birmingham Medical College, aged 72, died May 23rd.

Dr. George A. S. Ryerson, Niagara-on-the-Lake, Ontario, Canada, aged 70, died at Wellesley Hospital, May 20th. He was emeritus professor of ophthalmology and otology, University of Toronto.

### SOCIETIES.

At a recent meeting of the New York Ophthalmological Society the following officers were elected: President, Dr. Henry H. Tyson; vice-president, Dr. W. G. Reynolds; secretary and treasurer, Dr. John H. Dunnington.

The Scott County and Rock Island County Medical Societies held a joint meeting at Davenport, Iowa, May 4th, following a dinner. Dr. W. F. Boiler, professor of ophthalmology State University of Iowa College of Medicine, spoke on the "Problem of Lighting and Illumination as It Affects the General Practitioner."

The officers elected for 1925, of the Section on Ophthalmology of the American Medical Association are as follows: Chairman, Dr. Arnold Knapp, New York; vice-chairman, Dr. Harry Friedenwald, Baltimore, Md.; delegate to the house of delegates, Dr. William H. Wilder, Chicago; alternate, Dr. Harry S. Gradle, Chicago; member of the American Board of Ophthalmic Examinations, Dr. E. C. Ellett, Memphis, Tenn.

### PERSONAL.

Dr. Wm. Ford Blake of San Francisco is vacationing in the Hawaiian Islands.

Dr. Henry A. Beaudoux of Minneapolis, Minnesota, will locate in Oakland, California.

Dr. L. D. Green of San Francisco is taking a rest in the Hawaiian Islands.

Dr. Otto Barkan is spending the summer in northern Europe and will attend the Ophthalmological Congress in London.

Dr. William D. Rowland has been promoted to the grade of associate professor of ophthalmology in the School of Medicine, Boston University.

Dr. Richard J. Tivnen, professor of diseases of the eye, ear, nose and throat, Loyola University School of Medicine, Chicago, received the honorary degree of doctor of laws at that university, June 9th.

Dr. W. H. Wilmer, Washington, D. C., sailed for Europe, June 2nd, to survey research methods. He will visit Paris, Vienna, Berlin, Breslau, Freiburg, Leipzig, London, Glasgow and Edinburgh, to study late laboratory methods in ophthalmology.

Dr. David Harrower of Worcester, Massachusetts, was recently elected president of the American Ophthalmological Society; Dr. Wil-

liam Zentmayer, Philadelphia, vice-president; and Dr. Emory Hill, Richmond, Virginia, secretary-treasurer.

Dr. George W. Jean of Santa Barbara will attend the Eye Congress in London in July and after that he and Mrs. Jean will motor through France and Spain, returning to California in September.

Doctors William M. Perdue, James D. Perdue, and Colvin C. Perdue of Mobile, Alabama, announce that beginning June 1, 1925, their practice will be limited as follows: James D. Perdue, the eye, and Colvin C. Perdue, the ear, nose and throat.

Dr. William F. Brownell of Fort Collins, Colorado, announces that after June 1st Dr. Louis Packard will be associated with him and that Dr. Packard will continue the practice during Dr. Brownell's absence abroad.

Dr. William F. Boiler, professor of ophthalmology, State University of Iowa College of Medicine, has resigned and Dr. Cecil S. O'Brien, formerly of the Wills Eye Hospital staff, Philadelphia, has been appointed to succeed him. Dr. Boiler has been associated with the University eighteen years. He will now engage in private practice.

Dr. George E. deSchweinitz of Philadelphia will leave for Europe late in June to attend the English Speaking Congress of Ophthalmology in London July 13th to 17th. He will later represent the American Medical Association at Bath, England, as delegate to the British Medical Association and will return to Philadelphia about the middle of October.

Dr. William M. Sweet of Philadelphia, clinical professor of Ophthalmology of Jefferson Medical College, has been elected to the professorship of ophthalmology to succeed Dr. Howard Forde Hansell who recently resigned after a distinguished career of thirty-one years in association with the Jefferson School of Medicine. Dr. Sweet's notable work as writer and teacher, as well as his association with the American Ophthalmological Society as secretary and president, makes this honor a fitting climax to his career.

### MISCELLANEOUS.

The Eye, Ear, Nose and Throat Hospital, New Orleans, received a bequest of $5,000 from T. H. McCarthy, one of the trustees.

Under the will of the late Margarette E. Griffith, the Manhattan Eye, Ear and Throat Hospital, New York, received $5,000.

The old home of the Brooklyn Eye and Ear Hospital has been sold to William A. White, who will present it to the Polytechnic Institute. The Brooklyn Eye and Ear Hospital will erect a new building at a cost of $1,500,000.

The Trachoma Hospital at Pikeville, Kentucky, which has been maintained by the United States Public Health Service for about ten years, has been ordered closed. It appears that this hospital has been doing an excellent work and always had a long waiting list and that this condition exists at the present time. The community furnished the building free of cost to the federal government and recently has spent $600 in repairs at the request of the Public Health Service.

Professor L. Koeppe of the University of Halle, Germany, recently gave a course in Slitlamp Microscopy to a group of Cleveland ophthalmologists. The course was given at the Lakeside Hospital and extended over a period of twelve days. It was the fifteenth course that Professor Koeppe has given in the past year in the United States. Beginning Septmber 15th, the professor will again lecture in New York City at the New York Eye and Ear Infirmary. Following the completion of that course he will be ready to give further demonstrations and lectures in other large cities where arrangements may be made for them. He has also received invitations to give courses in Canada, Mexico and Brazil.

### THE WILMER INSTITUTE.

On February 15th last, President Goodnow of Johns Hopkins University announced the completion of the William Holland Wilmer Foundation of a $3,000,000 fund for the establishment at Johns Hopkins of a great clinical institute for research, teaching and treatment in the field of ophthalmology. The foundation of the Wilmer Institute is regarded as a matter of great importance to Johns Hopkins and to this country. Dr. Wilmer took his degree in medicine at the University of Virginia with the class of 1885. After serving as interne at Mount Sinai Hospital in New York and having conducted a course of lectures on ophthalmic surgery in the Polyclinic Hospital of New York, he began the practice of medicine in Washington, D. C., in the year 1889, where he has since resided. Thru his great industry and conscientious work, coupled with a marked ability and a kindly spirit to all classes of persons he has been extremely successful in his chosen specialty, and in the course of a comparatively short span of years he has become the leading ophthalmologist of this country. In recognition of the great service which he has rendered to his fellowmen and as a fitting culmination to a truly great career, the Wilmer foundation was organized.

The idea of this foundation very appropriately originated in the mind of a lady who was one of his patients, he had saved her sight, and while she was resting in the hospital and recuperating from the effects of an operation, she learned of the tremendous research still to be done in connection with diseases of the eye and of the fact that there were not adequate funds available in America or abroad for carrying out systematic research work.

So, in December, 1922, a group of friends and patients of Dr. Wilmer organized the William Holland Wilmer Foundation under the laws of the District of Columbia. The purpose of the original foundation was to raise one and a half million dollars for the establishment and endowment in the city of Washington of a center for the care, treatment and surgery of the eye. Washington was selected because of Dr. Wilmer's associa-

tions there and because of the fact that in Washington he had been forced to do his work with inadequate facilities in an institution which was dependent for its support upon a very limited field. It is particularly interesting to note at this point that the original articles of incorporation provided, among other things, that the foundation was "created as a tribute to the character, professional attainment and service to humanity" of Dr. Wilmer, and that "among its purposes are the organization, preservation and extension of the tradition of scientific skill and unselfish service to humanity maintained by Dr. Wilmer in his chosen specialty and the advancement by any suitable means of that field of science and practice wherein he worked." This is indeed a worthy tribute to any man, and one of which the University of Virginia as his alma mater may well be proud.

The trustees of the foundation chosen were as follows: Robert W. Kelley of New York, who became president; Henry Breckinridge, also of New York, who became secretary and treasurer; Mrs. Aida de Acosta Root, Mrs. Douglas Robinson, Felix Warburg, Mrs. William K. Vanderbilt, jr., Mrs. William Ross Proctor, Herbert L. Satterlee, and David Dow, all of New York; Robert L. Thompson, of Southampton, L. I.; William P. Eno, of Washington; Joseph Pulitzer, of St. Louis; Mrs. Henry R. Rea, of Pittsburgh; Major General Merritt W. Ireland, of Washington, surgeon general of the army; Harold F. Pierce, assistant professor of Physiology, Columbia University; and Walter B. Howe, of Washington, D. C.

The work of soliciting the necessary funds progressed slowly until November, 1924, when the board had collected about $200,000 thru private subscription. At that time it was found that the trustees of Johns Hopkins University had in mind a similar establishment in their program of the endowment campaign for the university and hospital. Realizing that with the advantages of such an institution Dr. Wilmer's influence would be broadened into one of national and international importance, the trustees of the Wilmer foundation decided to join forces with the Johns Hopkins trustees in erecting, equipping and endowing a suitable institution, which it was estimated would require about $3,000,000. $1,000,000 of this amount was to be expended for the buildings and equipment, $1,000,000 for the endowment of beds and the treatment of patients, and $1,000,000 for the endowment of research and teaching. The plan was to complete this fund and then have the Wilmer Institute become a component part of the medical school at Johns Hopkins in association with the other services already established there.

The raising of so large a fund appeared to be an almost impossible undertaking and one which might take many years, but fortunately on November 20, 1924, the General Education Board of the Rockefeller foundation came forward and pledged one-half of the needed $3,000,000 on the condition that the other half be raised by private subscription on or before February 15, 1925. Even then the task was quite stupendous, particularly in view of the time limitation and the fact that no access could be obtained to Dr. Wilmer's case files; but a leading citizen of New York City is credited with saying some years ago in reference to the need for a certain institution: "If the man could be found, the money would be." The man was the essential factor; for an institution, as Emerson has aptly said, is but the lengthened shadow of a man. The fact that the right man in this case has been found was abundantly confirmed; for, promptly on February 15, 1925, Dr. Frank Goodnow, president of the Johns Hopkins University, announced that the fund had been completed and the greatest teaching hospital and research institute for diseases of the eye in this country and the only one of its kind in the world would be established immediately at Johns Hopkins and named the Wilmer Institute. Dr. Wilmer, he announced, was appointed director of the institute, professor of ophthalmology in the medical school, and ophthalmologist-in-chief to the Johns Hopkins Hospital.

Among the largest subscribers to the fund were: J. P. Morgan, G. F. Baker, and G. F. Baker, jr., Robert W. Kelley, Frank Munsey, Julius Rosenwald, Felix Warburg, James N. Hill, Ira Copley, Mrs. Henry R. Rea, and Clarence Mackay; but the most gratifying feature was the great number of very small subscriptions from grateful patients who were not in financial condition to make larger ones. The trustees of the Johns Hopkins Hospital also assigned to the institute a building and land valued at $200,000. The building, which was formerly a nurses' home facing on Broadway and adjoining the main group of buildings at Johns Hopkins, is now being enlarged and reconstructed so as to accommodate forty beds, and it is expected that it will be ready in time for Dr. Wilmer to begin his new work in the fall.

In spite of his busy professional career, during which he had as patients many of the most prominent men in this country and also many foreign diplomats, Dr. Wilmer has been quite active in the alumni affairs of the University of Virginia. During the war he was officer-in-charge of the Medical Research Laboratory of the Army Air Service at Mineola, Long Island, and was later surgeon-in-charge of the Medical Research Laboratories of the Air Service in the American Expeditionary Forces. For his work in these positions he was awarded the Distinguished Service Medal and was made a commander of the Legion of Honor by the French government. At the present time he holds the rank of brigadier general, Medical Reserve Corps, United States Army. He is also a member of numerous medical and honorary societies and has contributed many most valuable articles on the subject of ophthalmology.—*University of Virginia Alumni News.*

# Current Literature

These are the titles of papers bearing on ophthalmology. They are given in English, some modified to indicate more clearly their subjects. They are grouped under appropriate heads, and in each group arranged alphabetically, usually by the author's name in **heavy-faced type.** The abbreviations mean: (Ill.) illustrated; (Pl.) plates; (Col. Pl.) colored plates. Abst. shows it is in an abstract of the original article. (Bibl.) mean bibliography and (Dis.) discussion published with a paper.

## BOOKS

American Academy of Ophthalmology and Oto Laryngology. Trans. 25th annual meeting. Cloth, 572 p., 110 ills., 1 col. pl. Published by the Academy. Chicago. A. J. O., 1925, v. 8, p. 502.

**Lang, B.** Routine examination of the eye. Cloth, 12 mo., 156 p., 30 ills. London, Edwin Arnold and Co. New York, Longmans, Green and Co., 1925. A. J. O., 1925, v. 8, pp. 501-502.

Principles adopted for electric lighting in H. M. Naval Service, Admiralty Electric Lighting Committee. 51 p., 12 diagrams. London: H. M. Stationery Office. B. J. O. 1925, v. 9, pp. 314-315.

Public Health Service of the United States. Cloth, octavo, 310 p., illustrated. Government printing office. A. J. O., 1925, v. 8, p. 503.

**Siegrist, A.** Refraktion und Akkommodation des menschlichen Auges. 8 vo., 154 p., 108 ills. Berlin: Julius Springer, 1925. B. J. O., 1925, v. 9, p. 316.

**Wick, W.** Funktionsprüfung des Auges mit ihren anhang. Physiologie und Pathologie der Pupille. 146 p., 90 ills. Berlin: S. Karger, 1924. B. J. O., 1925, v. 9, p. 316.

## DIAGNOSIS.

**Flemming, P.** Significance of ocular symptoms. Lancet, May 23, 1925, pp. 1065-1067-1071.

**Gala, A.** Stereoscopic photography of the eye. Časopis lékarů ceskych, 1925, v. 64, p. 649. J. A. M. A., 1925, v. 84, p. 1972.

**Jackson, E.** Estimating vision in opacities of the media. A. J. O., 1925, v. 8, p. 500.

**Koby, F. E.** Optics and ocular microscopy. (4 ills.) Arch. d'Opht., 1925, v. 42, pp. 280-284.

**Marsh, E. J.** Clinical microscopy of the living eye. Jour. Med. Soc. New Jersey, 1925, v. 22, pp. 195-200.

**Peter, L. C.** Slitlamp studies of clinical cases. A. J. O., 1925, v. 8, pp. 491-492.

**Uribe Troncoso, M.** Gonioscopy and its clinical applications. (21 col. ills., 14 ills., bibl.) A. J. O., 1925, v. 8, pp. 433-449.

## THERAPEUTICS.

**Bailliart, P. and Terson.** Ophthalmic instruments. (6 ills.) Soc. d'Opht. de Paris, 1925, April, pp. 182-188.

**Bürger, M.** Insulin in ocular therapeutics. Zeit. f. Augenh., 1925, v. 55, pp. 326-342.

**Cowley, R. H.** Milk injections in eye infections. Kentucky Med. Jour., 1925, v. 23, pp. 270-272.

**Liebermann, L.** Ektebin in ocular therapeutics. Klin. M. f. Augenh., 1925, v. 55, p. 363.

**Malone, J. Y.** Scopolamin anesthesia in ophthalmic surgery. Southern Med. and Surg., 1925, v. 87, pp. 285-287.

**Pesme, P.** Butellin a local anesthetic. Soc. d'Opht. de Paris, 1925, April, pp. 188-190.

## REFRACTION.

**Ferree, C. E. and Rand, G.** Apparatus for quick and accurate location of the meridians of astigmatism. (2 ills.) A. J. O., 1925, v. 8, pp. 453-456.

**Genet, L.** Paralysis of accommodation in lethargic encephalitis. Clin. Opht., 1925, v. 29, p. 307.

**Milian.** Myopia from neoarsphenamin. Bull. et mém. Soc. Méd. des Hôp. de Paris, Dec. 6, 1924, v. 48, p. 1765. Abst., Arch. of Dermatol. and Syphil., 1925, v. 11, p. 832.

**Post, L.** Paresis of accommodation following toxin-antitoxin. A. J. O., 1925, v. 8, pp. 478-488.

**Reid, A. C.** Changes in refraction in diabetes mellitus. B. J. O., 1925, v. 9, pp. 317-318.

## OCULAR MOVEMENTS.

**Behr, C.** Diagnosis of abducens paralysis. (2 ills.) Zeit. f. Augenh., 1925, v. 55, pp. 293-308.

**Berens, C.** Red toric lens with handle for studying diplopia. (1 ill.) A. J. O., 1925, v. 8, pp. 485-486.

**Cushman, B.** Family of squinters. (Dis.) A. J. O., 1925, v. 8, pp. 494-495.

**Dean, F. W.** Tucking operation for convergent strabismus. A. J. O., 1925, v. 8, p. 493.

**Dohme.** Congenital oculomotor paralysis. Zeit. f. Augenh., 1925, v. 55, p. 344.

**Fergus, A. F.** Miners' nystagmus. Lancet, May 23, 1925, p. 1068.

Treatment of convergent concomitant squint. Brit. Med. Jour., 1925, May 30, pp. 1002-1004.

**Hester, J. H.** Tendon transplantation of eye muscle. Kentucky Med. Jour., 1925, v. 23, pp. 265-266.

Optical treatment of convergent squint Lancet, June 6, 1925, p. 1193.

**Pandy, K.** Amaurotic asynkinesis (Insufficiency of convergence.) Zeit. f. Augenh., 1925, v. 55, p. 363.

**Reese, W. S.** Operation for convergent squint. (Dis.) A. J. O., 1925, v. 8, p. 492.

## CONJUNCTIVA.

**Gifford, S. R.** Eosinophilia and etiology of Parinaud's conjunctivitis. (1 ill., bibl.) A. J. O., 1925, v. 8, pp. 450-462.

**Gutzeit.** "Targesin" (silver) in conjunctival inflammation. Deut. med. Woch., 1925, p. 659.

**Hillegas, W. M.** Trachoma. Hahnemannian, Mo., 1925, v. 60, pp. 362-367.

**Maiden, S. D.** Ophthalmia neonatorum. Perforation of corneas. A. J. O., 1925, v. 8, p. 493.

**Martin, A.** Tularemia infection of conjunctiva. Southwestern Med., 1925, v. 9, p. 232.

**Molloy, L. P.** Trachoma. Kentucky Med. Jour., 1925, v. 23, pp. 272-273.

**Paderstein.** Swimming bath conjunctivitis. (Dis.) Zeit. f. Augenh., 1925, v. 55, p. 345.

**Petres, J.** Psoriasis of conjunctiva. Zeit. f. Augenh., 1925, v. 55, p. 363.

**Shahan, W. E.** Neuropathic conjunctivitis. A. J. O., 1925, v. 8, pp. 461-463.

**Zentmayer, W.** Kuhnt-Heisrath operation for trachoma. A. J. O., 1925, v. 8, p. 491.

## CORNEA AND SCLERA.

**Cazalis.** Miotics in superficial keratitis and simple ulcers. Clin. Opht., 1925, v. 29, pp. 253-258.

**Gradle, H. S.** Transient ring opacity of cornea. A. J. O., 1925, v. 8, pp. 483-484.

**Joel, E.** Arcus senilis in young subjects. Arch. d'Opht., 1925, v. 42, p. 311.

**Lampert.** Pathology of cornea studied with slitlamp. (2 ills.) Arch. d'Opht., 1925, v. 42, p. 285.

**McCollum, E. V., Simmonds, N. and Becker, J. E.** Ophthalmia in rats produced with diets containing vitamin A and excess of salts. Jour. Biolog. Chem., 1925, v. 64, pp. 161-181.

**Pesme, P.** Hereditary specific interstitial and parenchymatous keratitis studied with biomicroscopy. (4 ills., 1 col. pl.) Arch. d'Opht., 1925, v. 42, pp. 225-232.

**Post, L. T.** Thermophore treatment of ring ulcer of cornea. (dis.) A. J. O., 1925, v. 8, pp. 486-487.

**Rollet and Colrat.** Corneal complications in exophthalmic goiter. Arch. d'Opht., 1925, v. 42, p. 302.

**Suker, G. F.** Corneal ulcers. A. J. O., 1925, v. 8, p. 496.

## ANTERIOR CHAMBER AND PUPIL.

**Mawas and Vincent.** Alkalinity of aqueous Soc. d'Opht. de Paris, 1925, April, pp. 190-192.

**Pfingst, A. O.** Pupil in diagnosis. Kentucky Med. Jour., 1925, v. 23, pp. 282-287.

**Seidel.** Origin of and changes in the aqueous humor. Deut. med. Woch., 1925, v. 51, pp. 851-855.

## THE UVEAL TRACT.

**Bonnamo and Genet.** Suppurating iridochoroiditis. Arch. d'Opht., 1925, v. 42, p. 250.

**Charpentier.** Syphilitic iritis after novarsenobenzol treatment. Soc. d'Opht. de Paris, 1925, April, pp. 172-178.

**Martin, H. H.** Visual field in toxic uveitis. (9 ills., bibl., dis.) Southern Med. Jour., 1925, v. 18, pp. 426-429.

**Simpson, J. H.** Iritis. Kentucky Med. Jour., 1925, v. 23, p. 321.

**Thompson, J. W.** Tuberculous keratoiritis. Colorado Med., 1925, v. 22, pp. 232-237.

## GLAUCOMA.

**Agnantis, C.** Visual fields in secondary glaucoma. Soc. d'Opht. de Paris, 1925, April, p. 196.

**Guist, G.** Inflammatory orbital processes and glaucoma. (3 ills.) Zeit. f. Augenh., 1925, v. 55, pp. 308-318.

**Pesme, P. and Parinaud, F.** Operative treatment of glaucoma. (bibl.) Arch. d'Opht., 1925, v. 42, pp. 289-298.

## THE CRYSTALLINE LENS.

**Adams, D. R.** The crystalline lens. (3 tables, bibl.) B. J. O., 1925, v. 9, pp. 281-299.

**Bagot.** Barraquer's cataract operation. Clin. Opht., 1925, v. 29, pp. 258-263.

**Calhoun, F. P.** Contraindications for operation of senile cataracts. Kentucky Med. Jour., 1925, v. 23, pp. 274-279.

**Cruickshank, M. M.** Choroidal hemorrhage following cataract extraction. (bibl.) Indian Med. Gaz., 1925, v. 60, pp. 213-217.

**Dean, F. W.** Mercury cyanid injections in cataract. (dis.) A. J. O., 1925, v. 8, p. 494.

Bilateral cataracts, macular degeneration. A. J. O., 1925, v. 8, p. 493.

Suction instrument for extraction of cataract. (ill.) A. J. O., 1925, v. 8, pp. 484-485.

**Findlay, E. K.** Congenital membranous cataract. A. J. O., 1925, v. 8, p. 216. Dis. p. 495.

**Jackson, E.** Traction and suction apparatus for cataract extraction. A. J. O., 1925, v. 8, p. 499.

**Jocqs, R.** Extraction of cataract. Clin. Opht., 1925, v. 29, pp. 249-253.

**Labbe, H. and Lavagna, F.** Chemical constitution of normal and pathologic lens. Acad. des Scien., April 14, 1925.

**Liebermann, L.** Wound treatment in cataract operation. Zeit. f. Augenh., 1925, v. 55, p. 362.

## THE VITREOUS HUMOR.

**Bailliart, P.** Ossification of vitreous. Soc. d'Opht. de Paris, 1925, April, p. 182.

**Scarlett, H. W.** New blood vessel formation in vitreous. A. J. O., 1925, v. 8, p. 493.

## THE RETINA.

**Bonnet, P.** Spasm of retinal artery. Soc d'Opht. de Paris, 1925, April, pp. 192-194.

**Crocco, A.** Lymph extravasation in retina (Purtscher's disease). Semana Med., 1925, v. 32, pp. 1002-1007.

**Deutsch.** Pseudoalbuminuric neuroretinitis. Zeit. f. Augenh., 1925, v. 55, p. 351.

**Fridericia, L. S. and Holm, E.** Night blindness and malnutrition. Amer. Jour. Phys., 1925, v. 73, pp. 63-79.

**Holm, E.** Hemeralopia in rats nourished on food devoid of fat soluble A-Vitamin. Amer. Jour. Phys., 1925, v. 73, pp. 79-85.

**Redslob.** Cortical blindness with eclampsia. Arch. d'Opht., 1925, v. 42, p. 307.

**Rieger.** Tapeto retinal degeneration. Zeit. f. Augenh., 1925, v. 55, p. 346.

**Russ, S. and Lawson, A.** Susceptibility of nocturnal animals to ultraviolet radiation.

(1 ill., dis.) Pro. Royal Soc. Med., Sec. on Opht., 1925, v. 18, pp. 37-42.

**Scarlett, H. W.** Nontraumatic holes of the fundus. (1 ill., bibl.) A. J. O., 1925, v. 8, pp. 467-470.

**Weber, F. P.** Retinitis pigmentosa and Recklinghausen's disease. Med. Press and Circ., May 27, 1925, pp. 416-419.

### TOXIC AMBLYOPIAS.

**Aub, J. C., Fairhall, L. T., Minst, A. S. and Reznikoff, P.** Ocular lesions in lead poisoning. Médicine, 1925, Feb.-May, v. 4, p. 202.

### THE OPTIC NERVE.

**Botey, R.** Posterior sinuses and retrobulbar neuritis. Arch. de Oft. Hisp.-Amer., 1925, v. 25, pp. 305-322.

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